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CONTRIBUTORS TO THIS NUMBER

- ISAAC A. ABT, M. D., Professor of Diseases of Children, Northwestern University Medical School; Attending Physician, Michael Reese Hospital (Sarah Morris Memorial Hospital for Children), Chicago.
- PETER BASSOE, M. D., Associate Professor of Nervous and Mental Diseases, Rush Medical College, Chicago; Attending Neurologist, Presbyterian Hospital, Chicago.
- ARTHUR F. BYFIELD, M. D., Associate in Medicine, Northwestern University Medical School; Attending Physician, Cook County Hospital, Chicago, etc.
- JAMES G. CARR, M. D., Assistant Professor of Medicine, Northwestern University Medical School; Assistant Professor of Clinical Medicine, Post-Graduate Medical School, Chicago; Attending Physician, Mercy and Cook County Hospitals, Chicago.
- GEORGE F. DICK, M. D., Assistant Professor of Pathology, Rush Medical College, Chicago; Pathologist, St. Luke's Hospital, Chicago.
- CLIFFORD G. GRULEE, M. D., Assistant Professor of Pediatrics, Rush Medical College, Chicago; Attending Pediatricist, Presbyterian Hospital and Home for Destitute Crippled Children, Chicago; Consulting Pediatricist, Provident and City Contagious Hospitals, Chicago.
- WALTER W. HAMBURGER, M. D., Associate Professor of Medicine, Rush Medical College, Chicago; Attending Physician, Cook County Hospital, Chicago; Associate Attending Physician, Michael Reese Hospital, Chicago; Director, Department of Medicine, Michael Reese Dispensary, Chicago.
- RALPH C. HAMILL, M. D., Assistant Professor of Nervous and Mental Diseases, Northwestern University Medical School; Visiting Neurologist, Wesley and Cook County Hospitals, Chicago.
- JULIUS H. HESS, M. D., Professor of Diseases of Children, College of Medicine, University of Illinois; Attending Pediatrician, Cook County, Michael Reese and Englewood Hospitals.
- MILTON M. PORTIS, M. D., Assistant Professor of Medicine, Rush Medical College, Chicago; Attending Physician, Cook County Hospital, Chicago.
- W. D. SANSUM, M. D., Associate in Medicine, Rush Medical College, Chicago; Assistant in Medicine, Staff of the Presbyterian Hospital, Chicago; Laboratory for Clinical Research, Chicago, etc.
- ROBERT SONNENSCHN, M. D., Assistant Professor, Oto-laryngology, Rush Medical College, Chicago; Professor Oto-laryngology, Post-graduate Medical School, Chicago; Attending Oto-laryngologist, Cook County Hospital, Chicago.
- SOLOMON STROUSE, M. D., Assistant Professor of Medicine, Northwestern University Medical School; Acting Attending Physician, Michael Reese Hospital, Chicago.
- FREDERICK TICE, M. D., Professor of Medicine and Head of Department, College of Medicine, University of Illinois; Member of Attending Staff, Cook County Hospital, Chicago.
- RICHARD J. TIVNEN, M. D., Instructor in Ophthalmology, Northwestern University Medical School; Attending Oculist and Aurist to Mercy Hospital, Chicago; Consulting Oculist and Aurist, Provident Hospital, Chicago, and Illinois Central Railroad.
- CHARLES SPENCER WILLIAMSON, M. D., Professor of Medicine, College of Medicine, University of Illinois; Attending Physician, Cook County Hospital, Chicago.
- FRANK WRIGHT, M. D., Assistant Professor of Clinical and Physiological Chemistry, Northwestern University Medical School; Attending Physician, Mercy Hospital, Chicago; Associate Attending Physician, Michael Reese Hospital, Chicago.

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THE MEDICAL CLINICS OF NORTH AMERICA

VOLUME 3

NUMBER 1

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL
FOR CHILDREN)

PROGNOSIS OF DISEASE IN INFANCY AND CHILD- HOOD

ACCURATE prognosis is one of the most valuable assets of the medical practitioner. By forecasting correctly he inspires his patient, as well as his colleagues, with confidence. Too often the prognostic factor is assumed to be self-evident, and the medical curriculum as well as the medical literature fails to give this subject the emphasis which its importance merits.

The physician is expected to make a statement concerning the nature and gravity of the disease from which the patient is suffering. In those instances where he cannot reach a definite conclusion he gains the respect of patients by stating his own limitations. Spectacular prognoses of the hour of recovery and prophesying the onset of a measles rash after Koplik spots have appeared may impress the layman at the time. Such prognoses are attended with considerable risk, and when they prove wrong the physician comes to occupy the unenviable position of a false prophet. Who has not heard the lament of grief-stricken parents: "Doctor, you did not know our child was so sick! You did not expect this disaster." On the other hand, the physician who has made a fatal prognosis feels humiliated, to say the least, when the patient has recovered.

The general principles underlying prognosis are the same in children as in adults. An accurate prognosis as to the outcome

in a given disease can only be made when the diagnosis is definitely known and the underlying pathologic processes are understood. Hippocrates, the father of medicine, considered prognosis extensively, but his prognostications were made without accurate diagnosis and without pathologic knowledge. His rules of practice were based on experience. He was, as his critics have said, "the physician of experience and common sense." With particular attention he studied the natural history of diseases, that is to say, their tendencies to a favorable or fatal issue; and "without knowledge what can all medical practice be but blind empiricism."

Prognosis is a changing condition from age to age. The mortality of certain diseases of childhood is not as high as it was fifty or one hundred years ago, or even ten years ago because of the marked advance in therapeutics. Diphtheria and cerebrospinal meningitis need only be mentioned, though it is true that late complications are frequent because a greater number of patients survive as the result of better treatment. Improvement in hygiene, the advance in pathologic and etiologic knowledge of disease, and the consequent application of the principles of prophylaxis have diminished the incidence of morbidity as well as mortality.

A difference in life expectancy certainly exists during the various periods of infancy and childhood. Thus, if we compare mortality of the acute infections during the first five years of life, we find that fatal results are more frequent in the first and second years than during subsequent years. The infant possesses a certain degree of immunity by inheritance, but, on the whole, the resistance to acute infections is less developed than among older persons and adults. We explain this by saying that his antibodies are only feebly developed. For example, a newborn baby who falls ill with erysipelas is nearly always lost. An older child usually recovers. We dread to encounter scarlet fever in an infant, as he is likely to develop a malignant type of the disease or numerous complications as a result of general sepsis so that the outlook is extremely unfavorable. Sepsis of the newborn infant occurs easily after local infection, and death is the rule. Inju-

ries to the head of the newborn baby are likely to result in meningeal hemorrhage, which offers an unfavorable prognosis either to life or health of the patient, resulting in death or idiocy and paralysis. A tuberculous infection in a young baby tends to present itself as a disseminated miliary type with manifestations of fever, marasmus, and usually terminates fatally. In contradistinction to this, older children may acquire tuberculous processes of the bones, joints, or glands, but the process tends to respect its localization, for a time at least.

There is another element in prognosis which should not be overlooked. I refer particularly to the personal element reflecting the thought, the experience, the so-called clinical intuition, and individuality of the physician himself. The personal equation varies among physicians. Some are natural optimists. They believe in the goodness and restorative power of nature. They believe in themselves and their ability to cure. They bring cheer to the patient and hope to his friends, and, in reality, they lighten the burden of disease. The optimist who delivers himself of opinions which are not based upon experience and accurate knowledge of disease, however, will have a high percentage of failures.

The pessimistic physician who forecasts gloom, who diagnoses the most serious conditions which could occur under the circumstances, who pictures the remotest complications, represents the extreme type. We leave the charlatan entirely out of consideration. Assuming the diagnosis to be correct, we must give credit to the pessimist for emphasizing the variable possibilities of disease. His pessimism may be born of an overemphasis of the pathologic processes viewed at the autopsy table. Perhaps he is haunted by the spectre of perverted chemical processes and the possibilities of bacterial invasion. He has lived in the hospital and ministered to the sick, where they were brought as a last resort. The after-image has remained as a dark coloring to his prognoses.

It goes without saying that accurate prognosis cannot depend upon the personal equation of the medical man, be he optimist or be he pessimist. The majority of physicians find it easier to give

a favorable prognosis if consistent with the findings because of the satisfaction it gives.

The attitude of the parents is not to be disregarded. The attitude toward life and death is not always the same. Every human attribute is represented among our patients. Some are stoics; some are fortified by religion and faith. Some wish to be apprised of the exact truth. The sensitive natures who are so highly organized emotionally require to be protected against harsh expressions. Extreme tact must be employed, so we conclude that prognosis must needs be influenced by the varying mental attitudes of the parents as well as physicians' tendencies.

In so far as prognosis can be accurate, it reflects the wisdom, the thoughtfulness, the experience, and the knowledge of the medical attendant. By his wisdom he reflects the quality of his mental processes, his power of reasoning. He collects all the facts and is enabled to give a logical opinion, but he does not lose sight of the human element in prognosis. He must not unnecessarily depress the patient or his friends. Likewise, he must not pronounce a cruel sentence upon the patient when there is some uncertainty or a reasonable doubt as to the outcome. He should give cheer to the patient and hope to the family when it is possible. He will attempt to protect his own reputation for sagacity, and, above all else, he will endeavor to communicate to the parents the absolute unmitigated truth as he understands it. A wise doctor upholds the reputation of his calling and maintains unsullied his character as a man.

By his thoughtfulness he divests himself of all preconceived opinions and all personal relationship. He seats himself by the bedside of his patient and makes the minutest investigation that is possible in a clinical way. He invokes the aid of the laboratory and establishes the diagnosis on the basis of which his prognosis should be founded.

By virtue of his knowledge of the world of medical experience, his personal experience, and the information obtained at the bedside of his patient he estimates the prognostic values as they apply to his patient in particular.

After the diagnosis has been stated the parents ask, What is

the immediate outlook, and what are the possibilities of complete restoration? Let us pause for a moment and consider. Is a definite diagnosis possible? What is the constitutional endowment? What hereditary influences may be at work? What complications are present or impending? In our reply we are confronted with the question whether it is justifiable at times to be evasive or to make considerable reservations so as to permit escape from subsequent reproach. Is a physician in duty bound to utter the literal truth as he sees it?

Every right-thinking man will agree that there should be no escape from telling the unvarnished truth, but it is not necessary to communicate fears which may not materialize. How frequently everyone of us has been in error in diagnosis as well as prognosis! It must necessarily occur that accurate prognosis, even when the diagnosis is known, cannot always be stated. Who knows what the morrow will bring forth? A typhoid fever patient is apparently progressing favorably, and in three or four days he has developed a perforation or fatal hemorrhage. Who could have foreseen it? A diphtheria patient succumbs to cardiac failure. A convalescent scarlet fever patient develops a fatal nephritis in the third or fourth week. These are the mitigating circumstances which make a definite prognosis difficult or impossible. An older colleague consulted with me one night at the bedside of a child who was seriously ill. In the presence of very anxious parents we agreed on the diagnosis and recognized the danger of the situation. He spoke words of wisdom, however, when he advised me never to make a bad prognosis at night on the first visit to a patient at the onset of an acute illness.

"Things may look different in the morning," he said. "Since danger is not impending, let one or both of these parents have a night's sleep. They will be steeled for whatever may happen."

We spoke words of cheer. It happened as the doctor presaged. The child showed less prostration the following day. The meningeal symptoms which she presented proved to be the prodromes of a severe pneumonia which ran a favorable course. Obviously, this rule does not always apply. If an acute surgical emergency exists, and a fatal issue is even remotely possible, no

right-thinking man would defer his opinion for a moment. As a general principle of prognosis, to defer an unfavorable opinion until morning for further reflection and study is the part of wisdom.

The older clinicians considered their prognosis under two heads—the prognosis *ad vitam* and the prognosis *ad valetudinem*. These are the points concerning which the parents ask to be satisfied. The prognosis as to life depends somewhat upon the time the patient is examined. At the onset of a disease the symptoms may be mild or severe, and the outlook is conjectural. Nevertheless, certain important points are considered at this period. What is the average fatality of this disease under the best treatment which can possibly be employed? What is the general constitution of the patient? Is he robust or is he enfeebled by previous disease? Was he breast or artificially fed? It is a well-known clinical fact that breast-fed infants not only thrive better but show greater resistance against infections than bottle babies. Moro showed that the blood of breast-fed infants, even if weak and delicate, has greater bactericidal power than that of the artificially fed, even if healthy. Has he been wisely managed, or is he suffering from the effects of ignorance, poverty, or neglect? In a word, has he lived under hygienic conditions? The family physician often excels the specialist in prognostication by having a full knowledge of the previous history and the reactions of the patient.

Heredity plays an important part in estimating the prognosis. Some children, like some adults, seem to be composed of good material. Wear and tear and rough usage seem to have slight effect. Other children are composed of poor material. They do not seem able to withstand the insults to which they are subjected. They crumble, fade, or wilt under maltreatment. This composition of tissue, cell, and organ, this weave of the goods of which they are made, betrays the stock from which they come. These reflections should have an important bearing on prognostication. The severity of the infection must not be lost sight of. Some children seem doomed from the time they fall ill. Take the instance of a case of malignant scarlet fever. The child

is stricken in the midst of his play, perhaps on the street, is carried to his home, and never regains consciousness. Death ensues shortly. In a large group of cases, however, this does not apply. The onset of the disease is moderate, the severity increases from day to day, unexpected complications occur, and death may ensue. Epidemic diseases differ in severity from season to season and decade to decade. The virulence varies with the epidemic. Hirsch, the medical historian and geographer, states that mortality in scarlet fever varies from 3 to 5 per cent. in some epidemics, to 30 per cent. in others.

If we are dealing with an acute disease the prognosis may be changed favorably or unfavorably during its course. When complications occur and other organs than the primary seat become involved, when normal breathing changes to dyspnea, when cyanosis replaces the normal color of the skin, when the heart tones become rapid or feeble, or when cardiac dilatation occurs—or, indeed, when any vital organ begins to show symptoms which the trained observer interprets as unfavorable—the prognosis will naturally change as the ominous symptoms occur. The outcome may be death, or the health may become permanently impaired. We have already put the question, Is evasion justifiable? Are we in duty bound to express the literal truth? These questions cannot be answered in a word. In those cases where the physician is close to the family, where he is their confidant and helps bear their burdens, he will not find it necessary to communicate his exact fears at once. It is sometimes better that the parents themselves should come to a gradual realization of the problem which they have to confront. This applies particularly in the case of mentally defective children. The physician does not feel called upon to reveal the nature of the affliction at once, although the parents may be blind to the condition. In cases where parents suspect the true condition it may be well to defer a positive statement concerning the future mental life of the child. It is another matter where the family consults a specialist who does not enter intimately into the life of the family as does their own physician. Inasmuch as he sees the patient but once, no other course is left open to the specialist than to state candidly the

nature of the disease and the prospects for the future. The intense maternal love frequently prevents the mother from seeing the defects in her own offspring. She learns the truth gradually; consequently, her grief is less acute.

There is a variation in predisposition to disease at various ages, just as there is among various races. The difference in predisposition must depend upon variations in the structure of the body in its widest sense. There are undoubtedly differences between the structure of the infantile and adult organisms. A trauma inflicted on an infant or child is likely to result more seriously than an injury of equal force inflicted upon an adult. The younger the child, the more delicate its structure and thinner the cranial bones; thus, a blow on the head, which in a ten-year-old child might be without effect, may produce fatal results in an infant.

In a particular way the newborn infant is subjected to mechanical injury during birth. In severe labor the cranial sutures can be loosened, particularly if there has been instrumental interference during birth. In a general way it may be said that infants and young children are more easily and seriously affected by mechanical insults than older children or adults. During infantile life the child is, for the most part, protected against external insults by the nursing care of the mother, but when it begins to be more independent in the world it begins to be exposed to trauma. The surgeon sees more traumatic conditions in children between the sixth and twelfth years than he does in infancy and young childhood.

The possibility of regeneration of tissue in children is more favorable than it is in adults. In this way some of the variations in the course of disease between children and adults may be explained. Wounds heal rapidly and easily in the young. In normal progress of growth and development the child presents the possibility of recovery from diseases which could not occur in an adult. Lobar pneumonia in young children runs a more favorable course and with a lower mortality than in later life. This may be explained in part by the fact that degenerative processes have not yet occurred in young life. The little patient has

not been vitiated by bad habits, such as alcoholism, overeating or overworking, or such pathologic conditions as arteriosclerosis, and myocardial and renal degenerations. On the other hand, children may have been damaged very early in life on account of syphilitic or alcoholic parents. The constitution of the child may be permanently weakened by malformations, constitutional defects, or congenital debility.

In infant asylums and infant hospitals children are not infrequently found dead in bed, where suspicion often rests on the attendants. Death may be preceded by a high temperature. Fatality frequently occurs from obscure causes where lethal results were not expected, as a result of congenital malformations, internal hemorrhages, or aspiration of food into the larynx. Status lymphaticus, likewise, is often the underlying explanation where death occurs from slight illnesses, administration of anesthetics, and the introduction of a tongue depressor. Closely associated with this is anaphylactic shock seen in older children.

The diseases of early infancy are due particularly to nutritional disturbances and diseases of the gastro-intestinal and respiratory tracts. Occasionally infectious diseases, such as occur in later childhood, may occur in infancy. How far one may speak of immunity in infancy against certain infectious diseases is difficult to formulate in the present state of our knowledge.

If we inquire briefly into the common diseases of childhood at the various periods of life we find that during the first year of life gastro-intestinal disorders, those conditions resulting from injuries at birth including congenital debility and premature birth and the diseases of the respiratory organs are most frequent.

The mortality during the first year of life is high, and its occurrence depends upon several well-recognized factors. First, the absence of maternal nursing; second, errors in diet, unhygienic care and nursing, and the evil effects of poor housing and bad air as they prevail among the poor.

In the second year of life the infectious diseases begin to play a more important part in the mortality lists. Diarrhea and enteritis still head the list, and bronchopneumonia comes in for a close

second. After this, measles, whooping-cough, diarrhea, croup, scarlet fever, and tuberculosis to a lesser degree.

In the third year of life diarrhea and enteritis are less frequent causes of death. Diphtheria and croup occur more frequently than bronchopneumonia, though this latter disease follows very closely on the heels of diphtheria and croup. Measles and whooping-cough during the third year of life contribute very largely to the mortality, measles causing 5.5. per cent. of deaths, whereas whooping-cough produces 4 per cent. of deaths.

In the fourth year of life diphtheria and croup head the list. Scarlet fever, bronchopneumonia, lobar pneumonia, diarrhea, and enteritis fall in about the same group, constituting between 7 and 7.5 per cent. of the deaths.

In the fifth year of life the statistics are about the same; bronchopneumonia and lobar pneumonia are less frequent causes of death than scarlet fever and tuberculosis. It is to be noted during this entire period that the typhoid fever death-rate has fallen very materially in the last decade.

From the tenth to the fourteenth year the mortality from measles and whooping-cough is no longer of prominence, though scarlet fever and diphtheria during this period give significant mortality figures. Between the tenth and fifteenth years scarlet fever accounts for 3 per cent. of the deaths and diphtheria for approximately 6 per cent. During this period pneumonia shows the lowest death-rate in any age period of life.

In this list of diseases I have briefly indicated the effects of age on prognosis during the various periods of infancy and childhood. I have intentionally avoided a detailed account of mortality statistics. However, valuable tables, classified according to age and disease, are readily accessible in the literature. It has been my desire rather to treat prognosis as it applies to the young individual in a general more than a specific way. In doing this I cannot avoid reflecting my own experience in the very difficult and uncertain art of medical prognostication. May I say that in reducing this theory of probabilities to percentage or to a mathematical basis the average of perfection attained by the medical man is none too high?

A wise prognostication must, first of all, be a well-trained physician. Prognosis does not occupy the same place in medical knowledge as pathology or diagnosis. As an art by itself it lacks precision. It depends on factors themselves variable. To be able to prognosticate the results of disease in infancy and childhood one must have a definite knowledge of disease, a wide experience, and a sound judgment. In the words of Hippocrates, "experience is fallacious, and judgment difficult."

A CASE OF HANOT'S CIRRHOSIS IN A TWO-YEAR-OLD CHILD

THE patient that I present to you this morning has been under our observation for more than four months. This little girl, who is at present two years old, was brought into our out-patient clinic about five months ago because, as the mother stated, she was losing in weight and suffering from a persistent and generalized jaundice. She was irritable and fretful and was growing progressively weaker. Previous to her coming to the clinic she was able to stand and walk, but now she is unable to do either. The mother said the child had been sick for about four weeks before being presented at the clinic.

On the following day she was admitted to the hospital. There was no additional information obtained from the mother except her observation that the sclerotic coats were becoming more intensely yellow. She also observed that the child had a good appetite, slept well, and that her bowels moved regularly. She thought that the child was slightly dyspneic. There is no history of previous illnesses. The family history shows that the father is living and well, and the mother herself is well, although not robust. There are three other children apparently normal. The mother affirms she has never had a miscarriage.

The child was admitted with a temperature of 98.6° F., which very soon rose to 100.4° F. The pulse varied between 100 and 120 and the weight was 15 pounds, 6 ounces. Examination showed a fairly well-developed white female child about twenty months old with a universal jaundice. The examination of the head and chest was negative. The abdominal examination showed that the liver was palpable 5 or 6 inches below the costal margin and that the liver area was increased to the left so that it was definitely palpable in the epigastric region and under the left costal arch. The spleen was palpable, large, hard,

and freely movable. As the history noted the scleræ were jaundiced and the skin was of a deep yellowish hue. The urine varied in color from a deep straw to a brownish green. The reaction was acid, and there was no albumin, sugar, or acetone. There were no casts or cells to be found. The urinary bile-tests were strongly positive. The feces showed some reaction for chemical blood, but bile was absent. The blood showed 16,000 leukocytes, 78 per cent. neutrophils, 18 lymphocytes, 4 large mononuclears, 80 per cent. hemoglobin, and 4,000,000 red cells. It was evident from the blood analysis that, aside from the slight leukocytosis with an increase in the neutrophil cells, no definite significance could be attached to the blood findings. The von Pirquet test was negative. The throat culture contained no diphtheria bacilli, and the vaginal smear was free from Neisserian organisms. The Wassermann test was negative.

The child is presented again today. You will notice that she is extremely wasted and markedly fretful. I again call your attention to the jaundice of her sclerotic coats. She weighs 17 pounds, 6 ounces today, and her temperature is 100° F. The jaundice is more intense than on the day she was taken from the hospital four months ago. It is to be emphasized that the jaundice has continued, and, if anything, it has increased. The color of her skin is more of a greenish than a yellowish hue at the present time. The liver is markedly hard and enlarged, although the surface is smooth, and the borders are plump and even. The spleen is large, hard, and movable. The examination of the urine shows that it is turbid and dark. It contains no sugar and only a little bile. The stools are hard and dry, although they apparently contain bile. We have then, to repeat, a white female child of two years who has been sick for five months with persistent jaundice, enlargement of the liver and spleen, a moderate ascites, and persistent wasting.

We have now arrived at the point where it is incumbent upon us to classify this case and to determine the clinical and pathologic nature of the disease. I may anticipate your own mental processes in analyzing the symptoms and course of the disease by saying that there is every reason to exclude a syphilitic hepa-

titis, though, of course, you know that syphilis is a common cause of inflammatory processes in the liver. The liver is not nodular as though there were syphilitic gummata. There is no evidence of syphilis in other parts of the body. There is no history of lues in the parents, and the Wassermann reaction is negative. The rarer forms of hepatic enlargements may be readily excluded. Malignant neoplasm, echinococcus cyst, passive congestion, fatty degeneration, such as occurs in intoxication and phosphorus-poisoning, amyloid disease, and abscess may be excluded by the general course and symptoms. Briefly, malignancy is characterized by nodular swellings, uneven surface, inequality of the lobes, more marked cachexia, and metastases. Echinococcus cyst is usually localized to one lobe or the other and is characterized by a rapid unilateral growth. Jaundice is the exception. The hard smooth edges of the liver are obscured, and exploratory puncture of the cyst reveals the characteristic hooklets. The blood shows an eosinophilia. In passive congestion there is a heart lesion to explain the swelling which is definitely absent here. It is obvious that we need not consider amyloid disease, abscess, or fatty degeneration. Banti's disease and a similar disease described under the caption of Gaucher's disease are characterized by splenomegaly. In fact, the spleen attains immense size, and the liver becomes moderately enlarged. The jaundice at present is not so intense. The blood findings, for the most part, show a leukopenia.

We have now discussed the case by excluding a great number of disorders which might possibly occur in the liver. We may say, however, that on account of the increasing size of the liver and the deepening jaundice this infant is suffering from a cirrhosis of the liver. Aside from luetic cirrhosis, which is of frequent occurrence in children, we know that two forms of cirrhosis may occur, namely, the atrophic, alcoholic or Laennec's cirrhosis, and the hypertrophic biliary cirrhosis, also called Hanot's cirrhosis. We have now to decide whether the little girl we are demonstrating this morning is to be considered a case of atrophic or Laennec's cirrhosis or one of hypertrophic biliary or Hanot's cirrhosis.

The atrophic cirrhosis has been frequently reported in young children. It is often due to abuse of alcohol in these young subjects. Ascites occurs early. After a brief stage of hypertrophy the liver contracts, and the surface and edge may be somewhat irregular on palpation. The superficial abdominal veins are dilated, and hemorrhages may occur from the mucous surfaces, such as the nose, stomach, and bowels. Jaundice is sometimes observed, but it is usually of a moderate degree. In the later stages of the disease ascites becomes very marked. The liver shrinks in size and very frequently is not palpable. Diarrheas are of frequent occurrence, and these children show increasing signs of weakness and prostration. Death takes place from some terminal infection. Such are the symptoms and course of an atrophic cirrhosis, but if we again refer to our patient we will note that the symptoms just related are not present in the little girl who is before us. Her liver has increased in size. The jaundice is persistent and progressively more intense. We feel that we are fully justified in making the diagnosis of hypertrophic biliary cirrhosis or Hanot's cirrhosis. It is true this is a rare condition. At least, it has not been frequently described.

The clinical features of the disease first clearly reported by Hanot in 1876 are those of a chronic form of jaundice accompanied by an enlargement of the liver and spleen and by intercurrent attacks of abdominal pain and pyrexia. Early ascites is infrequent. While the disease is not common in temperate zones, it has been frequently described in India and Mexico, where it is said to attack young children and even infants. Neither alcohol nor syphilis appear to bear any cause or relation in the cases that have been reported, though, on the other hand, the infective fevers, such as typhoid and scarlet fevers, seem to bear a close etiologic relationship. The symptoms which are usually noted have already been described, namely, general malaise, headache, abdominal pain, persistent jaundice, and a slight degree of pyrexia. Vomiting occurs at times. The liver is always enlarged, and the surface is smooth and firm. The spleen is enlarged and may attain a considerable size. The

jaundice is the earliest symptom and continues until the end. As in our case the urine contains bile pigment, and the stools are, for the most part, bile stained. The fever, to which reference has already been made, may occur early in the disease, though after a time it tends to disappear. There may be a slight leukocytosis which is most marked in the febrile case and which tends to persist. These patients sometimes show exacerbation which has already been referred to crises. During these attacks the liver and spleen may exhibit tenderness and may take on an additional enlargement. The crisis is of short duration, lasting only a few days, at the end of which time all the acute symptoms disappear, although the liver and spleen are larger than they were before the crisis. Clubbing of the fingers and deformities of the nails have been described as occurring in children. Arthritis has also been observed. Brown pigmentation of the skin resembling that of Addison's disease has been noted. These children develop slowly, and there is frequently a marked physical retardation. From the reported cases one concludes that the duration of hypertrophic biliary cirrhosis is variable. It may continue for several years, but, as a rule, the course of the disease is not so protracted as in adults and is marked by greater severity. The crises become more frequent, and emaciation and prostration become more marked. Hemorrhages from the skin and mucous surfaces may occur, and the scene is usually closed by coma resembling that which occurs in the severest forms of icterus.

The cause of this disease is obscure, but is probably due to some chronic form of infection. This view is made more probable by the recurring exacerbations or crises and the more or less continuous leukocytosis. This is further substantiated by the occurrence of a cholangitis or an inflammation of the smaller bile-ducts. The pathology consists of fine almost intercellular scarrings throughout the liver with an enlargement of the spleen and fever. Microscopically, there is a finely diffused network of scar tissue separating the lobules into little groups of cells.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

A MEDIASTINAL TUMOR

DURING the past few months a considerable number of medical cases have been presented in this clinic, some of which are of more than passing interest. Two have been selected for the purposes of this clinic.

The first patient, a female thirty-one years old, entered the hospital April 25, 1918, with the following complaint:

Onset and Course.—Patient stated that she had been sick for five weeks, with a cough and difficulty in breathing. With this she has had some difficulty in swallowing, particularly when she drank water, and if she takes a large amount and attempts to swallow it quickly. The symptoms have not changed much in the past five weeks. At no time have there been any acute exacerbations of her trouble.

Complaint in Detail.—*Cough* has been present, as stated, for five weeks, but during the first week it was very slight.

Expectoration was limited in amount, whitish in appearance, never hemorrhagic or foul smelling.

Dyspnea has been present continuously, and is more marked on exertion or when she lies down.

Dysphagia is manifest when she drinks water, and is particularly noticeable when she attempts to swallow hurriedly, which causes the water to return through the nose. On attempting to swallow solid food she complains that it sticks in the esophagus on a level with the upper part of the sternum. Solid food has never been regurgitated or vomited, nor has water failed to wash it down. Accompanying the difficulty in swallowing with the food lodging, there is frequently considerable discomfort, and at times actual pain.

Tumor.—No complaint was made of a swelling in the right supraclavicular area, but, upon questioning, the patient states that it has been present for some sixteen or eighteen months. When first noticed it was small, hardly noticeable, and has gradually increased in size.

Loss of Weight.—She does not believe she has lost any weight, with the possible exception of the last few weeks.

Pruritus has been present over the entire body for about eleven weeks, and is more pronounced on disrobing. It has been particularly marked during the past five weeks.

Appetite.—The patient's appetite is good.

Constipation is present, requiring frequent cathartics.

Past Illnesses.—Smallpox as a child.

Personal History.—Denies the use of alcohol, and gives a negative history for any venereal or specific infection.

Marital History.—First husband dead; cause of death unknown. Patient has given birth to 7 children, all living and well. No history of abortion or miscarriages.

Family history negative.

Physical Examination.—The patient is dyspneic and has to sit upright in bed; the face, lips, and hands are markedly cyanotic. On exertion shortness of breath is produced, followed by labored, noisy breathing.

Scattered over the entire body there are numerous traumatic lesions due to scratching. On the left side of the face in the region of the chin, as well as over the lumbar region, there are areas of vitiligo.

Chest.—The upper part of the sternum is more prominent, but no definite localized bulging can be determined. Expansion is poor, and on percussion the left border of the heart is outside of the left nipple line; the right border is approximately normal. Over the base of the heart, extending to either side of the sternum but more particularly to the right, there is an increased area of dulness. Palpation fails to reveal a pulsation or cardiac shock over this area of dulness. Auscultation reveals the presence of a soft, systolic murmur at the apex of the heart and one over the aortic area. No abnormal sounds can be

heard over the upper portion of the sternum. Auscultation over the pulmonary area, anterior and posterior, is difficult and inaccurate owing to the difficulty in breathing and the loud, noisy respiratory murmurs. In the right supraclavicular area there is present a mass about the size of a large hen's egg which is smooth and rather freely movable. This mass extends downward below the level of the clavicle. Several small glands can be palpated in the right cervical region. Moderately enlarged glands are present in both axillary and inguinal regions, as well as some enlargement of the epitrochlears.

Abdomen.—Examination of the abdomen is difficult, as the patient is unable to assume the recumbent position on account of her dyspnea, except for very brief periods of time. Percussion and palpation reveal an enlargement of the liver extending to three fingers below the costal border. The spleen is not palpable.

Head.—The head is negative except for slight dilatation of the right pupil and oral signs of a marked pyorrhea alveolaris.

Cerebrospinal examination negative.

Laboratory Findings.—*Urinalysis* negative.

Blood.—Hemoglobin, 80 per cent., by Sahli. Reds, 5,200,000; whites, 8600. Differential count, normal. Pressure: systolic, 110; diastolic, 70. Wassermann reaction negative.

Roentgenologic Findings.—The following is a report of the fluoroscopic examination: "A large mediastinal growth crowding the heart and aorta to the left. Outline of shadow is very distinct and does not pulsate."

While the patient was under observation the temperature never exceeded 99° F.; the pulse varied from normal to 120, and the respirations are recorded in the 20's and 30's.

Subsequent notes are to the effect that the patient suffered more or less continuously from dyspnea, amounting at times to orthopnea, cough, and difficulty in swallowing. At times these manifestations became more or less acute in the form of recurring paroxysms. She remained under observation for one month, at which time, owing to lack of improvement, she demanded her discharge, and left the hospital on the 25th

of May. She was under medical supervision at home, with her condition gradually becoming worse, and finally, on the 20th of June, she was induced to return to the hospital. At that time her cyanosis, dyspnea, cough, and difficulty in swallowing was most acute. The area of cardiomediastinal dulness had increased, the tumor in the neck had become larger, and there was an edema of the entire body, more marked in the right upper extremity and the right face and neck.

A fatal termination occurred in a severe attack of dyspnea about twenty-six hours after admission.

Discussion.—From a consideration of the symptoms and findings it was necessary to differentiate between several conditions, particularly a Hodgkin's, aortic aneurysm, and a mediastinal neoplasm. At no time was it possible to obtain permission to remove one of the glands for microscopic examination. While a Hodgkin's was one of the considerations, it was not sufficiently evident to justify such a diagnosis. Of the other possibilities mentioned, aortic aneurysm and mediastinal neoplasm, the following points were considered. Owing to the greater frequency of aneurysm it was considered necessary to give this first consideration, and the following points in favor of an aneurysm may be enumerated:

1. History of luetic infection.
2. Physical findings due to luetic infection, as tabes, periostitis, cardiohepatic involvement, etc.
3. History of repeated abortions or miscarriages.
4. Symptoms—anginoid pains more frequent.
5. Physical signs:
 - (a) Diastolic shock.
 - (b) Aneurysmal second sound—both important.
 - (c) Expansile pulsation—less important.
 - (d) Tracheal tug.
 - (e) Difference in peripheral pulse—carotid, subclavian, radial, etc.
 - (f) Regional lymph-glands not enlarged.
 - (g) Venous engorgement, collateral circulation, and cyanosis less frequent in aneurysm.

- (h) Murmur:
 - 1. Aneurysmal.
 - 2. Cardiac, particularly aortic-diastolic.
- 6. Roentgenologic:
 - (a) Fluoroscopic—expansile pulsating tumor in connection with artery or heart.
 - (b) Skiagraphic:
 - 1. Tumor shadow of aorta or heart.
 - 2. Outline definite and regular.
 - 3. No metastases in lungs, pleura, or bones.
- 7. Rapidity of development less in aneurysm.
- 8. Time element:

Duration greater than eighteen months, probably aneurysm.
- 9. Exploratory puncture in selected cases.
- 10. Possible influence of specific treatment.

This patient was admitted to the hospital on the clinical diagnosis of "mediastinal tumor and bronchitis." After observation, and with the findings as presented, a diagnosis was made of mediastinal lymphosarcoma.

Autopsy Report.—This will be given in abstract, and only the important and essential findings indicated. These were as follows: The head, neck, and ears are markedly cyanotic. Multiple areas of vitiligo about the chin and left cheek. Neck is short and thick; superficial venous trunks of neck markedly engorged. In right supraclavicular region there are several discrete, sharply circumscribed, enlarged glands, one about 5 cm. in diameter. Striking unilateral edema of right side of face, neck, chest, and right upper extremity. Right axillary glands moderately enlarged. On section, whole upper thorax is filled with one enormous solid tumor which displaced heart downward and to the left. No fluid in either pleural cavity or peritoneum. Liver enlarged. Description of tumor: In size about 16 cm. in diameter, fills entire mediastinal space, surrounding great vessels and adherent to pericardium. External surface irregular, but not nodular. Cut surface is smooth, with some bands of fibrous tissue. Opened pericardium reveals

mass about 4 to 5 cm. at base of heart anteriorly, which is rough and nodular. Growth is intrapericardial extension from mediastinal neoplasm. Heart is somewhat enlarged, but practically negative, except opened right auricle reveals mass about 3 to 4 cm. in diameter, similar in appearance to one in peri-

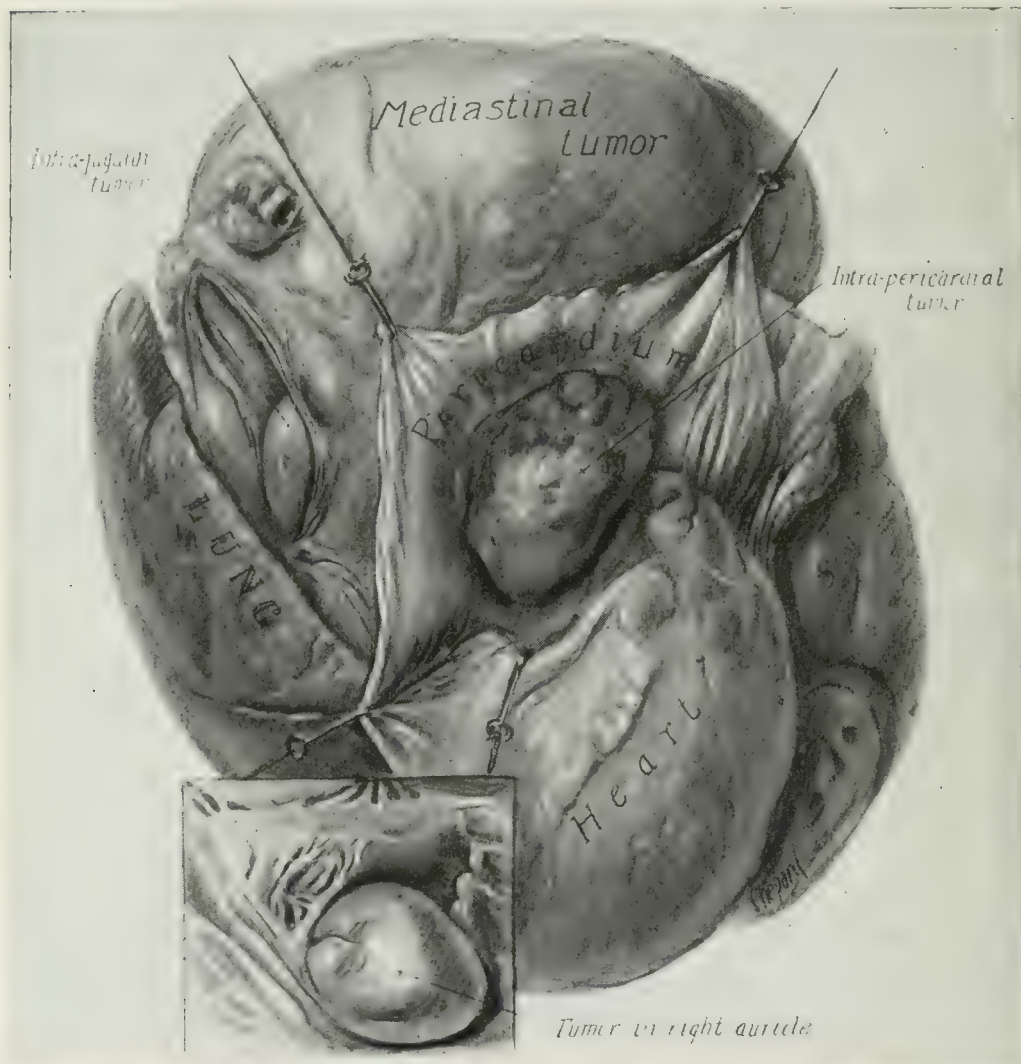


Fig. 1.—Anterior view, with large mediastinal neoplasm above, also showing growth into pericardium, right auricle, and right jugular vein.

cardium. Intra-auricular mass is extension from mediastinal growth, extending through wall of right auricle. On opening great vessels, right jugular is almost occluded by intravascular growth from primary neoplasm. (See Fig. 1.)

Microscopic Report.—Lymphosarcoma.

CARCINOMA OF THE STOMACH

THIS patient, a female seventy-one years old, entered the hospital March 23, 1918.

Present Illness.—*Discomfort and Pain.*—The patient is sure, that she was perfectly well up to three months ago, when she developed a dull, constant pain in the epigastrium, which was aggravated by eating solid food. Soft food and liquids would give her temporary relief. The discomfort and pain is present over the whole epigastrium, and radiates into the lower posterior thoracic region.

Loss of appetite appeared about the same time with the discomfort and pain. Due to the distress there is a fear of taking food, but aside from this there is an actual loss of appetite.

Vomiting appeared two and a half months ago, and was produced particularly by taking of any solid food, but might appear even with liquids. Eructations, nausea, and bitter taste would appear immediately on eating, which was followed in fifteen or twenty minutes by vomiting. She would often vomit during the night, the vomitus large in amount, and containing food particles which she had ingested the day before. The vomitus often contained blood, bitter in taste, but never sour. So far as can be elicited the vomitus has never had a "coffee-ground" appearance.

Loss of Weight.—During the past four months there has been a loss of 35 or 40 pounds.

Tumor Mass.—About one month ago the patient first noticed a tumor in the epigastric region in the area of the maximum discomfort. This has gradually increased in size.

Past History.—The usual diseases of childhood. No history can be elicited of previous gastric involvement or distress. No history of bowel hemorrhage.

Personal and family history negative for any bearing upon present condition.

Physical Examination.—*Abdomen.*—On inspection, there is seen a mass in the epigastrium which moves with respiration. Palpation reveals a very large, irregular, hard mass filling the greater part of the epigastrium, somewhat larger to the right side. This mass, while moving with respiration, is more or less fixed and apparently adherent to the liver, the lower border of which extends two or three fingers below the costal border. Several smaller, irregular masses in the neighborhood and surrounding the larger mass are also present. The gall-bladder cannot be palpated. On distending the stomach with gas moderate peristaltic waves are produced, appearing to the left and passing to the right. The soda bicarbonate and tartaric acid at once produced nausea, which was followed by vomiting and interfered with further observation. The vomitus contained food particles and blood.

Chest.—The lungs showed physical findings of an emphysema and bronchitis. The heart was slightly enlarged to the left, with a distinct systolic murmur at the apex. Heart and pulse-rate slightly increased, with a moderate degree of arrhythmia.

The remaining physical examination was negative except for a slight edema of the lower extremities and a small, irregular, firm mass about the size of a hazelnut located in the inner left supraclavicular region.

Laboratory Findings.—*Urinalysis* was negative.

Blood showed a moderate degree of secondary anemia.

Gastric Analysis.—A Weber on the vomitus showed the presence of blood. An Ewald was attempted, but was rather unsatisfactory, due to the nausea and vomiting. After the lapse of thirty minutes gastric contents were aspirated and gave a total acidity of 30 degrees, no free hydrochloric. Weber was positive.

Stool Examination.—A single specimen gave a positive Weber.

Roentgenologic Examination.—A motor barium meal was attempted, a portion of which was vomited, while the greater amount after six hours remained in the stomach. An extensive defect in the antrum and along the lesser curvature was present.

These findings were confirmed by a plate and presented the appearance of an extensive gastric carcinoma.

Discussion.—From the history, physical and laboratory findings, a clinical diagnosis was made of gastric carcinoma, with moderate grade pyloric obstruction.

Fatal termination occurred on the 31st, eight days after admission.

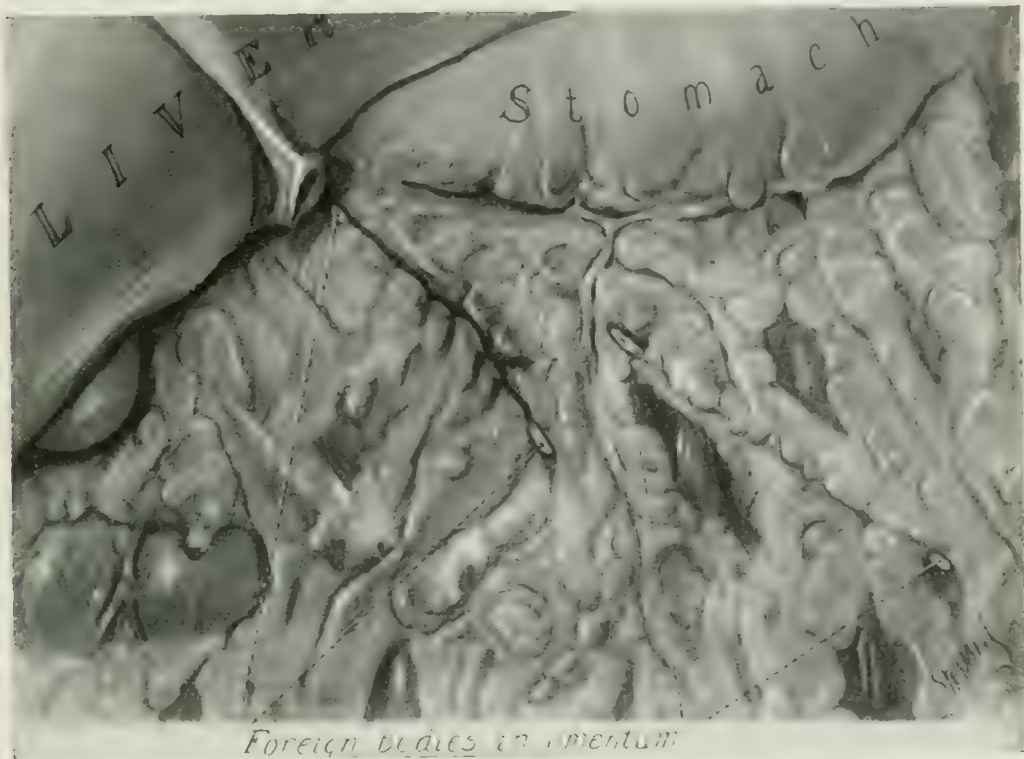


Fig. 2.—Anterior view of gastric carcinoma, giving location and position of foreign bodies.

Autopsy Findings.—The important abstracted findings were as follows:

Abdomen.—Peritoneum smooth and shiny. Under serous surface of small and large intestines there are multiple, white, small grayish nodules, which on section show a milky white, pus-like fluid. In the omentum in the region of the pyloric end of the stomach there are embedded and surrounded by adhesions two metal wires about the diameter of small knitting needles (Fig. 2). A large tumor mass is present about the pyloric end of the stom-

ach, involving stomach, liver, and omentum. In the gall-bladder there is a large solitary calculus. The stomach contains some fluid contents with putrefactive odor. In the region of the pylorus there is a huge ulcerating mass (Fig. 3) which has eroded through the stomach on the anterior surface; penetrating into the mass there is a metal wire about 4 inches long, placed per-

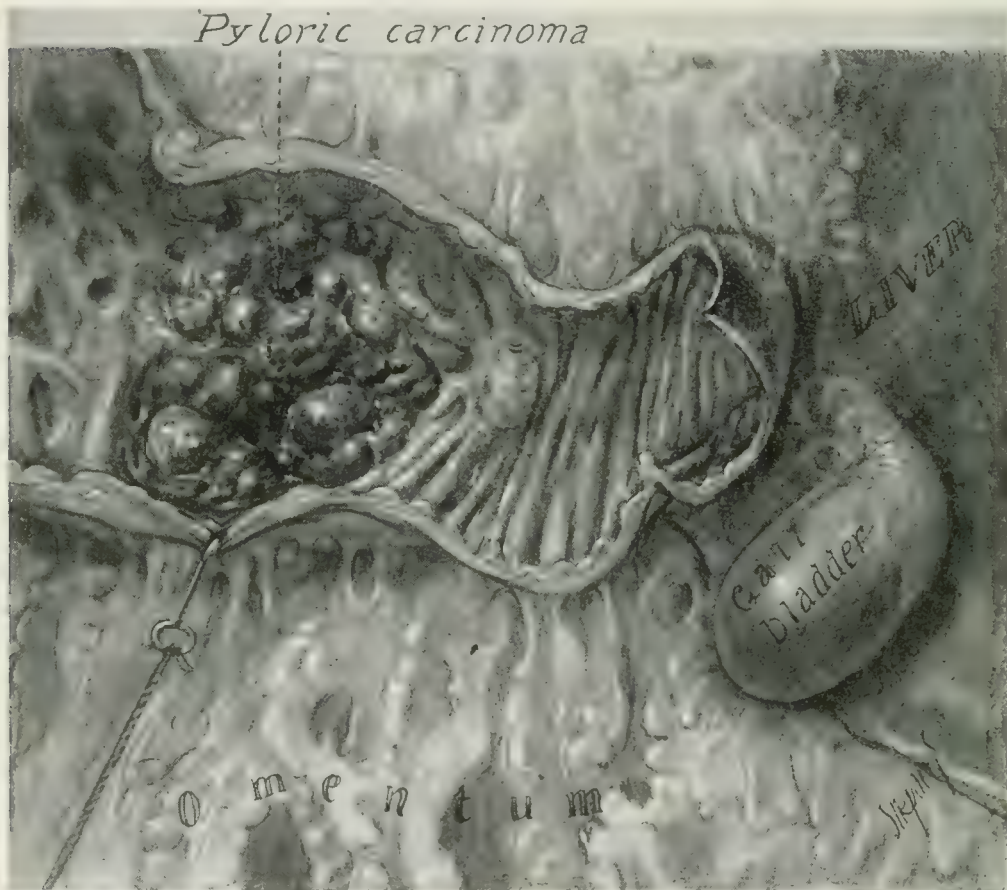


Fig. 3.—Posterior view of gastric carcinoma, stomach opened, exposing location of ulcerating carcinomatous mass.

pendicular and embedded in omentum and adhesions. Lying parallel and in close proximity a second wire is present and embedded in the omentum.

The remaining findings were non-essential, as may be judged from the anatomic diagnosis, which was as follows:

A huge chronic, destructive carcinoma of the pyloric end of the stomach and first portion of the duodenum; steel wires en-

capsulated in the omentum, fibrous adhesions between the duodenum, under surface of the left lobe of the liver, and about the gall-bladder. Multiple small metastatic growths in the peritoneum. Marked enlargement of the perigastric, biliary, retroperitoneal, and left cervical glands. Emphysema and myocarditis, fibroid type. (See Figs. 2, 3.)

Microscopic Sections.—Carcinoma.

While the clinical diagnosis of a gastric carcinoma was confirmed by the autopsy, the presence of foreign bodies was an unexpected finding. Re-examination of the Roentgen plate failed to reveal a shadow that could be interpreted as due to these bodies. Perhaps this could be explained as the plates were extremely unsatisfactory, partly due to defective technic and the inability to make good plates owing to the physical condition of the patient. So far as could be determined it would seem probable that the foreign bodies had been ingested, perforated, or escaped through a perforation of the stomach wall into the peritoneum. The deposit on the wire and the thoroughly embedded fibrous surrounding tissue would indicate that they had existed in the peritoneum for some time. While admitting the possibility that they may have gained entrance by some other route, particularly per uterus, it is less likely when we consider the gastric involvement and the negative pelvic findings. It seems reasonable to conclude that they were ingested and escaped through a gastric perforation.

CLINIC OF DR. JULIUS H. HESS

COOK COUNTY HOSPITAL

RADIOGRAPHIC DIFFERENTIAL DIAGNOSIS OF BONE AFFECTIONS IN INFANCY AND CHILDHOOD

THE patient presented here was admitted to the Cook County Hospital with the diagnosis of osteogenesis imperfecta tarda accompanied by hydrocephalus and secondary rachitic changes, with a complication of bronchopneumonia. There was no history obtained except that the child had always been ill. The father said that the enlargement of the head had been present from birth. The complaint at the time of entrance was loss in weight and poor health. Father's history so far as obtained was negative, the mother was sick with pneumonia at the time the child was admitted. There were five children, aged nine, eight, seven, and three years and one eight months old, all well. No feeding history was obtained. The mother had had four miscarriages.

Examination shows an emaciated child of about two years, with several bone deformities. The hair is lusterless, uneven, long, and fluffy. The head is hydrocephalic, square, with prominent parietal bones, and sweating. The eyes are bulging, pupils equal, and reacting to light and accommodation. The teeth are badly decayed, just appearing through the gums, there is also an inflammation extending around the entire course of the gums and tongue. The tongue presented many small spots of ulceration. The thorax shows a rosary on both sides anteriorly, the left thorax is larger than the right. The ribs are bulging on the left side posteriorly. There is kyphosis of the dorsal and lumbar regions, and lordosis of the upper dorsal region. There is pot belly, which is tympanitic, but not tender. All the

bones of the extremities are affected. Both femurs show thickening, probably the seat of old fractures, with a subsequent contraction of both legs and shortening of the hamstring tendons. There is a marked shortening of all the bones of the arms and legs. Reflexes are negative. Râles are heard throughout the chest, there being, however, no areas of consolidation and no bronchial breathing. The heart tones are rapid, but there are no murmurs. Urine is negative and Wassermann of blood negative.

In view of the history of repeated miscarriages on the part of the mother and the presence of ulcerative stomatitis and pain upon manipulation of the extremities of the child we do not feel justified in accepting the clinical diagnosis of osteogenesis imperfecta tarda with hydrocephalus and secondary rachitic changes, with a complicating broncho pneumonia, without due consideration of the possibilities of syphilis and scorbutus being present, notwithstanding the absence of blood in the urine and the negative blood Wassermann.

The **radiographic findings** show that the entire bony skeleton of this patient was involved in a high-grade osteo-atrophy; the long bones are very delicate in appearance, comparatively little distinction being noted between the cortex and the medulla. The former is, however, visible as a thin paper-like layer. The epiphyseal lines are (except the wrists) normal. Numerous intraperiosteal fractures are noted, the following bones being involved: left humerus three, left radius one, left ulna one, right humerus two, right ulna one, right radius one, right femur two, right fibula one, left femur one, a total of thirteen fractures. There is a peculiarity at the site of fractures noted which represents a ring of absorption of bone salts, but with definite callous formation surrounding these areas; the spine is seen deflected to the left in the middorsal region.

Normal Bones.—In examining the radiograms the following appearance of normal bones has to be kept in mind constantly: Periosteum is visible as a faint clear line, covering the surface of the bone. Beneath the periosteum compact bone (corticalis) is seen. Lamellæ may be sometimes demonstrated. Cancellous

tissue (spongiosa) in the center of the bone appears as porous network, the lamellæ appearing as irregular clear lines. Coursing through the interlamellar spaces blood-vessels may sometimes be made out. Epiphyseal cartilages in growing children appear as clear bands of varying depths passing across the outline of the bone. It is important to observe that the distal surface of the cartilage, that is, the surface lying next to the epiphysis, is smooth in outline, while the surface which looks toward the shaft, being the growing surface, is more irregular. Epiphyses lie beyond the epiphyseal cartilages, and, according to the age of the child, they may appear structureless, no ossification having developed, or they may show a small developing center of ossification, or may have structure similar to that of healthy cancellous tissue.

Osteogenesis Imperfecta Tarda.—The radiograms are invaluable in diagnosis and especially in the differential diagnosis of osteogenesis imperfecta, since the radiographic findings in this disease are characteristic and pathognomonic. They may be summed up as follows:

Multiple, mostly intraperiosteal fractures, often showing areas of bone resorption at the seat of fracture.

Excessive callous formation.

Deficient shadow formation seen in all bones of the body due to increased permeability to x-rays. Often the bone shadows show but little more density than the surrounding soft parts.

The diaphyses of long bones may be slender, and only very rarely show any curvature or bending.

The cortex is of irregular thickness, on the whole, very thin and parchment-like in appearance, and may even appear to be absent in some places. There is little or no tendency toward thickening on the concave side of the shaft.

The spongiosa contains wide meshes and an absence of structural markings. These changes are not limited to the diaphyses. All bones show this change, but not to the same degree, the most marked changes being found in the bones of the hands.

The medullary cavity is increased in size and shows irregularly mottled shadows.



Fig. 4.—Osteogenesis imperfecta.

The epiphyseal cartilages and their centers of ossification are larger than normal and the epiphyseal lines are straight (Figs. 4, 5).

Rachitis.—In rachitis the radiograms are not absolutely necessary to positive diagnosis, but in doubtful cases they are valuable for differential diagnosis. By radiograms it is possible to follow the process of healing of rachitis, to examine for fractures and infractions. In radiographic studies of bones we have the only means with which we may observe the effect of therapeutic measures.

Characteristic of the radiograms of the rachitic bones is the wide variation in appearance, not two radiograms being alike,

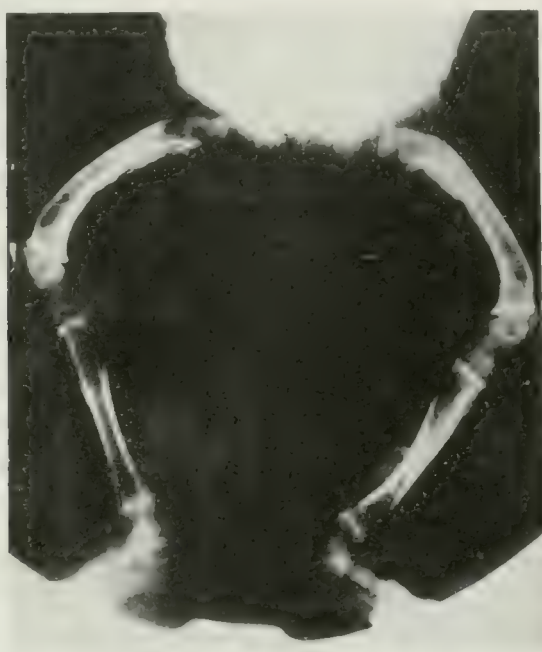


Fig. 5.—Osteogenesis imperfecta. Same case as Fig. 4, six months after treatment, showing marked improvement.

but in all pictures there are some common features which immediately stamp the bone changes as those due to rachitis.

The most important characteristic of rachitic bones in radiograms is their increased permeability to the rays due to diminished calcium content, this resulting in decreased intensity of the shadow and certain diminution of the contrast between the shadows of bones and those of soft parts surrounding the bones. The deficient shadow formation, of course, depends upon the degree of disturbance in calcium deposits, and therefore the intensity of the shadow is very variable.

In rachitis complete fractures are usual, but, as a rule, they are not intraperiosteal as in osteogenesis imperfecta, and there seems to be very little tendency toward callous formation, although apparently enough to make the bone more or less solid. The calcium content of the callus is so very low that it is almost invisible in a radiogram.

Deformities in rachitis are usually more marked in the lower than in the upper extremities. Bowing and bending is characteristic. In the femur the greatest change may be in the neck, causing coxa vara. The pelvis may become flattened and the body shortened as a whole.

After the process subsides the bones, while still showing deformities and curvatures, appear broader than normal, and show increased density of bone, which is especially marked at the ends of diaphyses.

The periosteum in rachitis is definitely thickened, but the periosteal bone shadow is often pale, due to the low calcium content.

The cortex is about the same in appearance as in osteogenesis imperfecta. On the concave sides of long bones, however, in rachitis the cortex is thickened, which is almost pathognomonic of rachitis. It may be thinned on the convex side.

The medullary cavity is often increased in size in rachitis and constantly so in osteogenesis imperfecta.

The epiphyses, which in osteogenesis imperfecta are often normal, show most marked changes in rachitis. The zone of proliferation is widened, with an irregular, serrated appearance on the epiphyseal side of the diaphysis, and there is also a broadening, flaring out, and cupping of the epiphyseal end of the diaphysis which is very characteristic. The epiphyses tend to be larger than normal, irregular in outline, and may be of decreased density. The end of the shaft is streaked and presents an irregular line at the joint end, and above it a curious transverse zone of different structure from the rest of the bone.

While the above findings are characteristic of a well-developed case of rachitis from the clinical standpoint, in completing our discussion the early or first stage and the later or third stage must

also be considered, as the early stage in severe cases more particularly offers considerable difficulty in differential diagnosis from osteogenesis imperfecta tarda.



Fig. 6.—Acute rickets. First stage, showing thickening of cortex, pale spongiosa, cupping and fraying of the ends of the shafts, with poorly developed epiphyses and haziness of joints.

First stage: Epiphysis casts little or no shadow, while the center of ossification is small or absent, and at times appears multiple. The diaphysis becomes frayed out, instead of clear cut, the periosteum thickened, and the whole joint appears

hazy. Multiple fractures are common. This is the stage which is frequently mistaken for osteogenesis imperfecta (Fig. 6).

Second stage: The shadow of the epiphysis becomes more marked, the area is ragged and irregular, the ends of the diaph-



Fig. 7.—Rickets. Second stage, showing marked thickening of periosteum, flared and saw-toothed appearance of ends of shafts, with increased bony formation in the nuclei.

ysis begin to broaden, especially on the side on which the strain is greater, and here produces a ledge or lip next to the epiphyseal line. There is a thickening on the concave side of the shaft which is compensatory in character. The diaphysis begins to

give a more definite shadow. The ends next to the diaphysis are streaked longitudinally and are the area of maximum disturbance. At the epiphyseal end of the shaft there is generally a transverse area of increased density, reaching about $\frac{1}{4}$ or $\frac{1}{2}$ inch from the epiphyseal line. This continues into the third stage. Further changes in the second stage consist in the chambering of the interior of the bone, where light areas in the shaft indicate the absence of marked bone deposit, and heavier lines of ossification show the irregular development of trabeculæ. The second stage is generally a period of systemic reaction to the disease, in which signs of returning ossification occur and when deformity begins (Fig. 7).

Third stage: The epiphysis begins to resume its normal contour and homogeneous shadow density. Irregularities persist in the marginal outline, and there is still a little mottling in the ossification. The lipping of the diaphyses has enlarged the bone ends, and there is, in consequence, a discrepancy in breadth between the diameters of the diaphysis near the epiphyseal line and the epiphysis.

Scorbutus.—Clinical diagnosis of a typical case is made on the basis of the following triad: (1) Hemorrhagic diathesis, which is manifested by tendency to hemorrhages and multiple hemorrhages anywhere in the body, with its sequelæ, marked anemia. (2) Severe pains in extremities accompanying every movement. (3) Swelling of the joints.

In scorbutus the radiograms are very valuable in establishing an early diagnosis at a time when there are no characteristic clinical signs of infantile scorbutus. There are also many rudimentary forms of the disease in which the only symptoms present may be moderate pallor, anorexia, and restlessness.

The bone changes are most frequent in the lower extremities and especially in the ends of the bones entering into the formation of the knee-joint. They occur somewhat less frequently in the distal portions of the bones of the forearm. They are of frequent occurrence in ribs at the place of junction of the bone with cartilages, and it is here where the bony changes may frequently be observed first.

The most constant finding in scorbutus is an irregularly circumscribed shadow of varying diameter and intensity at the end of the diaphysis at the seat of formation of new bone. This shadow, frequently described as "white line," enables one to make an early diagnosis of scurvy when all other classical signs of scorbutus are absent, provided it is possible to exclude congenital syphilis.

Zone of calcification at the junction of diaphysis with epiphysis is wider and more irregular than in syphilis, which it some-



Fig. 8.—Scorbutus. Hemorrhage into both knee-joints, with displacement of left epiphyseal nucleus. Also showing white line.

what resembles. The age incidence is always a valuable differential factor, osteochondritis syphilitica being the most common early bone finding in congenital syphilis, while scorbutus is rarely seen before the third month of life and most commonly later.

Less constant than this shadow are the subperiosteal shadows at the seat of hemorrhage, becoming more and more distinct with their duration, the increase in density being probably due to the deposit of osteophytes, which occurs in about two

weeks from the time of hemorrhage. The density of these shadows is usually intermediary between that of bone and that of soft tissues.

Fractures and infractions are demonstrable in scorbutus, but they are less common.

Other findings in scorbutus which, however, are among the less common findings are epiphyseal separations and displace-

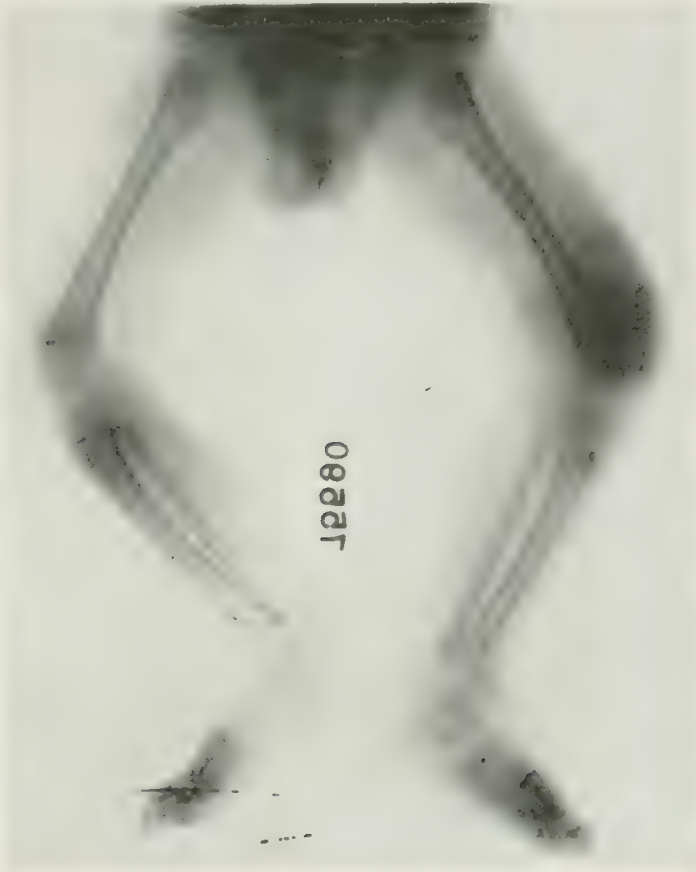


Fig. 9.—Scorbutus. Showing hemorrhage into left thigh, with beginning organization of hematoma and displacement of all of the lower ends of the diaphyses of the long bones.

ments, hemorrhages within the joint capsule, and occasionally, but rarely, intramuscular hemorrhages (Figs. 8 and 9).

Tuberculosis.—A destructive process is primarily and definitely characteristic of tuberculous lesions, without evidence of a tendency to bone stimulation, the destruction being always greater than new bone formation.

In general, in tuberculosis of the bone the increased permeability of the bone to rays is peculiar of this disease. The bone atrophy in tuberculosis is very early, and is apparent not only on the diseased bone itself but also in the immediate neighborhood of the focus. Not only the diseased portion of the bone but also the surrounding areas show in a radiogram a diminished shadow intensity, and therefore there is a lack of contrast in comparison to the soft parts.

Within the active zone of tuberculosis the corticalis of the bone is narrowed, the spongiosa appears as washed out. All around the active zone the bone appears enlarged, and when the focus is near the periphery the bones appear as if absorbed. Periosteal bony deposits are rather exceptions, the destructive process predominating.

Callous formation in tuberculosis is very deficient.

Frazer describes the radiographic appearance of a well-defined central disease as follows: An area in the medulla of the bone appears of diminished density, it corresponds to the tuberculous granulation tissue. Within the light area the lamellæ are absorbed, and around the periphery they are rarefied. There is an area well outside the diseased focus in which the lamellæ appears thickened. Between the lamellæ, around the periphery of the central deposit, the carpet of marrow loses its dark uniform appearance; it becomes lighter, and its structure has a suggestion of striation. This, which may be taken as a typical appearance, is altered under two conditions, namely, cavity formation and sequestration. When cavity formation occurs there is a diminution in resistance to the passing rays, and the area is registered as a dense black shadow, it may be in the center of the light tuberculous area. If a detached sequestrum is present it, as an object, offers a very considerable resistance to the rays. And in the radiogram it appears as an area somewhat lighter than that afforded by the diseased granulation tissue.

Peripheral changes are described by Frazer as follows: The earliest periosteal appearance is the development of what looks like a space between the periosteum and the underlying

bone. In reality it is the picture afforded by the first stage of the deposit of new subperiosteal bone granulation tissue, which has not yet become ossified. At a later examination there is a typical deposit of new bone between the periosteum and the underlying shaft. If the new bone is deposited upon the shaft it is cancellous; if it is laid down in the neighborhood of a joint



Fig. 10.—Tuberculosis of the bones. Erosion and dislocation of right femur.

its thickness is not so great, and its composition is comparatively denser. In examining an *x*-ray negative of new subperiosteal bone one should never omit to examine the surface of the underlying compact bone. In tuberculous disease such ought to be smooth. This is important, because it is a distinguishing feature between the appearances of a periosteal thickening due to underlying tubercle and that due to periosteal sarcoma. In the latter the outline of the compact bone is eaten out and irregular.

Tuberculous spina ventosa is to be diagnosed on the basis of preponderance of the destructive processes and at the same time lack of sclerotic processes. More marked shadow formation on the lateral edges of the diseased phalanges is usually sufficient for the diagnosis of the congenital syphilitic disease of the bone. Multiplicity of the lesions favors a diagnosis of syphilis, while when only one phalanx is involved the circumstance speaks

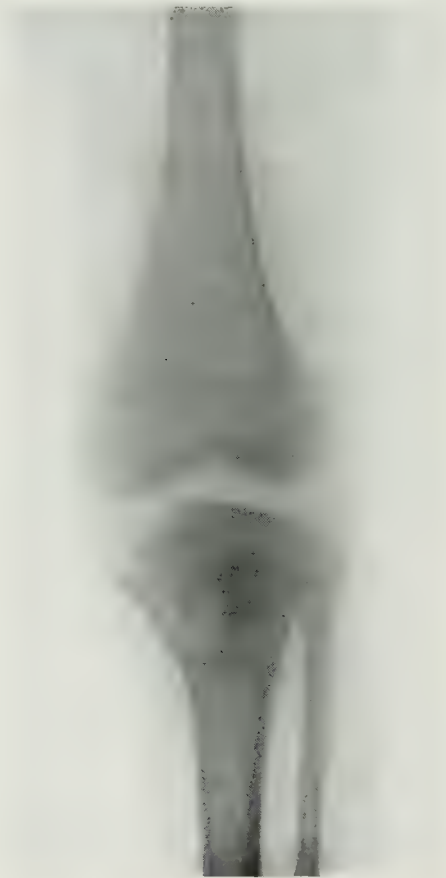


Fig. 11.—Osteomyelitis, acute, showing necrosis and sequestration in the upper end of diaphysis of left tibia.

rather for tuberculosis. However, syphilis should always be verified by the blood and spinal fluid reactions and therapeutic test (Fig. 10).

Osteomyelitis.—It is difficult to make positive diagnosis of osteomyelitis on the basis of radiograms before the end of the second week. By the second week we have evidence of a more or less marked periosteal proliferation and new periosteal bone

formation about the diseased area. Light and irregular shadows at the seat of the bone involvement are also now visible due to bone destruction. Later in the disease distinct bone sequestra surrounded by a definitely lighter shadow are characteristic.

Positive diagnosis of osteomyelitis is made on the demarcation of the sequestrum, on one hand, and unusually early and extensive ossificatory activity of the inflamed periosteum around the osteomyelitic focus. At the time at which the osteomyelitic focus in the bone due to purulent melting down of the bony substance is macroscopically visible in a radiogram as a light spot in otherwise normally dark bone shadow, there is also a fully developed and pretty extensive ossifying periostitis. The depth of the shadow produced by this periostitis is almost equal to that of the corticalis. This usually occurs in the second week of the disease.

In tuberculosis, on the other hand, periostitis of an ossificatory nature is, as a rule, absent. Thus in cases in which we see a larger lighter area in the shadow of the bone, without the least periosteal new bone formation, the latter appearance speaks strongly for tuberculosis. In osteomyelitis, sequestrum is usually definitely outlined, while in tuberculosis the line of demarcation is very indefinite (Fig. 11).

Chronic Osteomyelitis.—Typical radiogram of a case of chronic osteomyelitis in an advanced condition is as follows:

Areas of suppuration indicated by patches of varying density, rarefaction of the bone, and small collections of débris and pus.

Newly formed periosteal bone, shown by the deposition of successive layers of bone outside the shadow of the original bone or what remains of it.

Necrosis of the cortical bone, indicated by irregular patches of the denser shadow, with a well-defined periphery, beyond this being lighter shadows, where the living bone remains.

In general, these conditions are confined to the shaft of the bone involved, the epiphyses and joints escaping. The earliest radiographic manifestation is shown by a slight increase in the periosteal shadow at one or more spots, a definite swelling of the soft parts, and possibly an abscess (Fig. 12).



Sarcoma.—It is necessary to consider the appearance presented by sarcoma of the bone when dealing with what appears to be an inflammatory condition. Sarcoma usually attacks the shaft of the bone, and produces changes similar to those caused by certain degrees of osteomyelitis, differing, however, in that the latter shows a more pronounced degree of periosteal reaction as indicated by the deposition of new bone and the tendency to formation of sequestra. In medullary sarcoma certain areas of increased density appear which resemble spiculæ or islands of osseous material, and show actual absorption of the bone, with very few or no normal portions of the bone remaining about this point. In osteomyelitis, in addition to the more definite thickening of the periosteal shadow, there is more definite formation of new bone about the necrosed area.

Acute Infective Periostitis.—The diagnosis by radiogram of an early infection of the periosteum is obtained by noting changes, such as thickening and bulging of the periosteum. The outline of the periosteum in normal bone is sharply defined, while in acute inflammatory conditions there is a general haziness of its outline in the affected part, or it may be broken and irregular, exposing the cortex of the bone.

The formation of an abscess is shown by an increased depth of the shadow in the neighboring soft parts. In less acute cases this swelling may be due to inflammatory changes commencing in the periosteum. A careful study of the history and other signs of the case will avoid a mistaken diagnosis of scorbutus.

Syphilis.—Radiography is of great value in diagnosis of congenital syphilis. It allows us an insight into the condition of the skeletal system during life and an interpretation of the stage of the bone changes due to the disease. The radiographic pictures of bones in congenital syphilis vary widely, depending upon the intensity, stage, and localization of the pathologic changes.

In syphilis we must distinguish between the early congenital and the later bone changes. Characteristic of the early stage is

Fig. 12.—Osteomyelitis, chronic, showing necrosis and sequestration in left radius.

osteocondritis syphilitica, and of the later stage is periostitis ossificans.

Osteocondritis syphilitica is pathognomonic of syphilis, and may be found even in macerated syphilitic still-born infants in whom all other signs of syphilis may be absent. Radio-

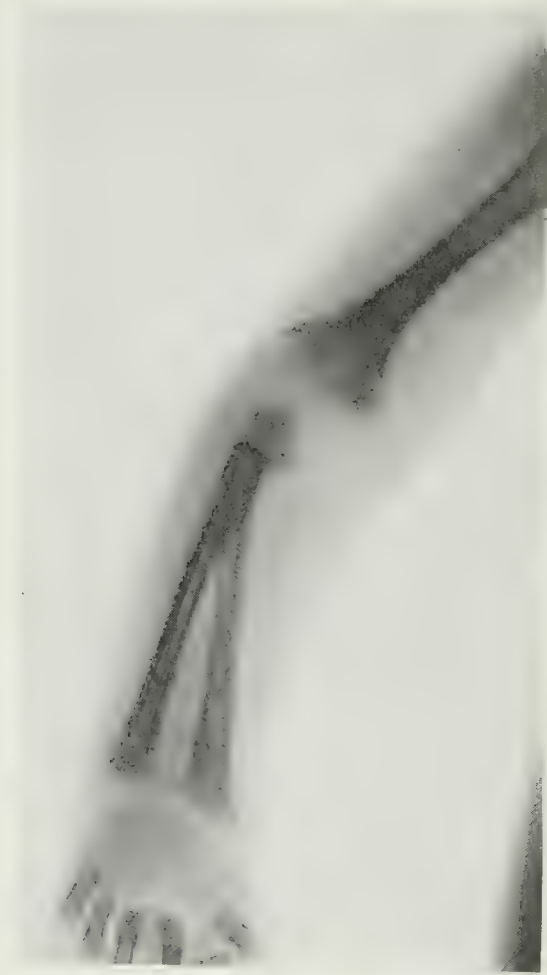


Fig. 13.—Early congenital syphilis, showing marked changes throughout the entire bone structure. Osteocondritis.

graphically it appears as either homogeneous transverse shadow or a streak interrupted by transverse streaks at the epiphyseal line, the latter being well outlined toward the diaphysis, and toward epiphysis a serrated ribbon-like shadow. The younger the infant, the more marked is the osteocondritis. In early congenital syphilis the long bones suffer chiefly (Fig. 13).

In later manifestations, in which periostitis ossificans is a very important finding, we see newly formed subperiosteal bony masses with bony structure. They are most marked in the middle of the diaphysis of the femur and tibia, and often present, but usually less marked in other long bones.

Periostitis ossificans produces deep shadows alongside the diaphysis parallel to the cortex and giving an appearance of double contour. Old cortex may often be differentiated from the newly formed subperiosteal bony masses. Cortical thickening is, therefore, an important point in differential diagnosis from osteogenesis imperfecta, in which disease the cortex is always very thin (Fig. 14).

In phalanges the epiphyseal line is somewhat lighter, the diaphysis is stronger and more compact, and forms a dark, sharply circumscribed shadow; the phalanges are longer and thickened. There are definite changes in the periosteum without any changes in the cortex and medulla, unless suppuration has taken place. Usually the lesions of the long bones are multiple, which is of assistance in differentiation of syphilitic dactylitis from the tuberculous spina ventosa.

Calcification zone which normally should be regular, slightly curved and narrow, appears in syphilis as a wider, irregular, serrated shadow of higher intensity. In the direction toward the diaphysis from the epiphysis one sees larger or smaller lighter areas due to presence of soft granulation tissue and deficiency or absence of bony trabeculae.

Diagnosis of gumma may be made with probability on the basis of presence of sharply circumscribed nodes of light spots.

In syphilis the extremities are chiefly affected. There may be changes in the ribs, but they are difficult of demonstration during life.

In making a differential diagnosis between syphilis and tuberculosis it must be remembered that the older the infant, the less pronounced becomes the osteochondritis syphilitica and the more marked becomes the periostitis ossificans, until in older children, in whom we speak of syphilis hereditaria tarda, the radiogram shows only the periostitis ossificans. This is

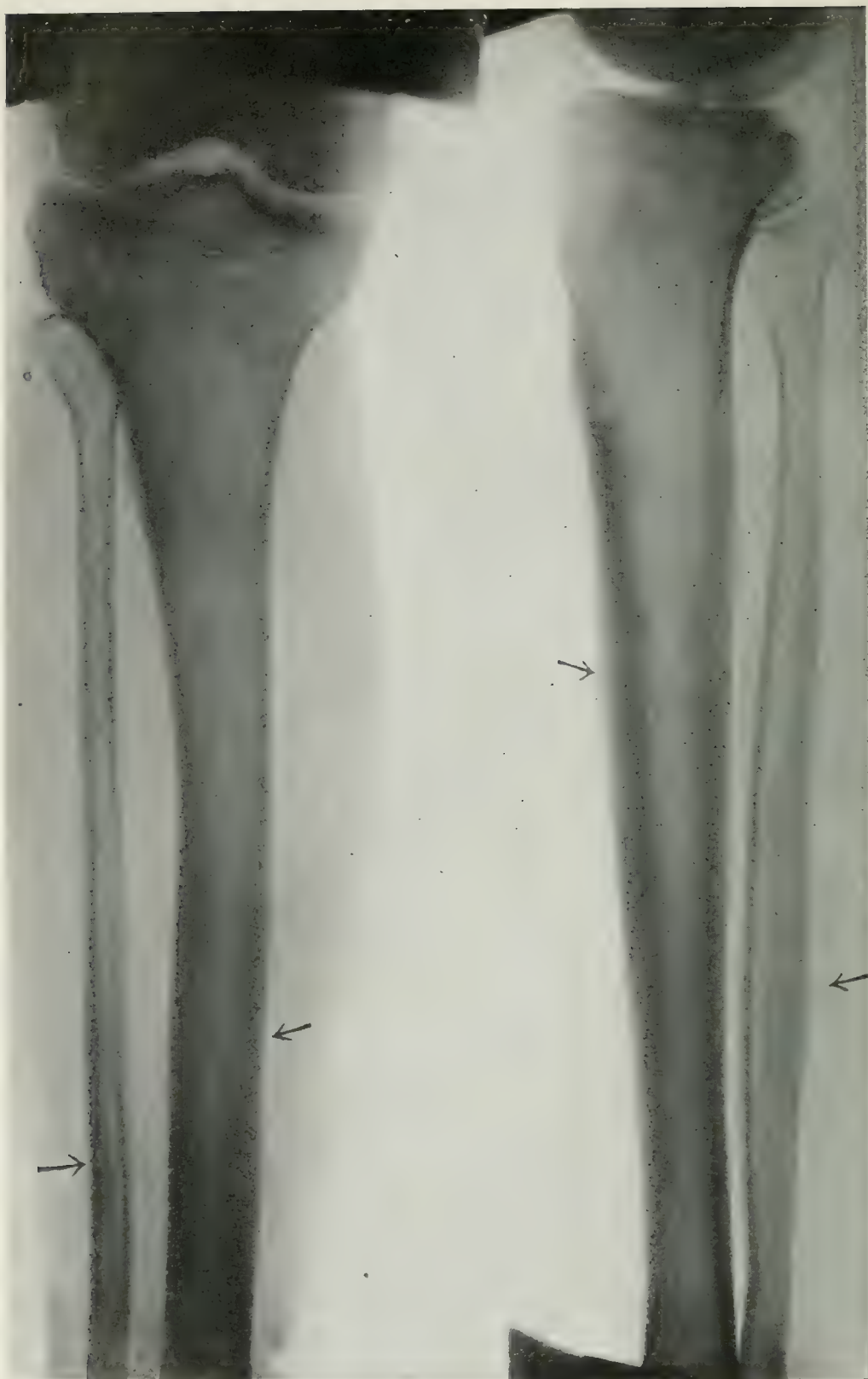


Fig. 14.—Congenital syphilis, with later bone changes of periostitis ossificans.

seen in the picture as an envelope of the diaphysis, often 1 millimeter wide or more, the longitudinal axis of the periosteal new bone formation being almost parallel to the long axis of the cortex of the bone. They may be confounded only with the involucrum of osteomyelitis and with some very rare cases of tuberculosis. The periosteal thickening in tuberculosis is rare, and when it is present it never produces shadows so dark as are those seen in congenital syphilis. In general, in the syphilitic periostitis the shadows are very deep, are more straight than curved, and thus may be used for differential diagnosis between syphilis and tuberculosis. Syphilitic periosteal processes are usually much more multiple than the tuberculous. In the tuberculous further not only in the region of the centers of ossification but also farther on in the diaphysis there is a high-grade atrophy of the bone.

It is more difficult to differentiate syphilitic periosteal changes from those occurring in simple osteomyelitis. Factors favoring diagnosis of syphilis are the multiplicity of the lesions, presence of osteochondritis syphilitica in infants. The outline of the periosteal deposits in osteomyelitis is somewhat more curved, while in syphilis it is more straight.

During the progress of healing of syphilitic bone lesions in a high-grade osteochondritic disturbance the pictures may be very similar to those of rachitis characterized by cupping. It is to be differentiated from rachitis by the fact that, comparing the two sides of the body, they show unequal involvement, while in rachitis the involvement is almost always uniform.

Periosteal sarcoma is differentiated by its destructive lesions involving the shaft of the bone as well as the cortical layers, and its limited tendency to periosteal thickening.

In reviewing the changes in radiograms produced by the different affections of the bones, the following changes may be regarded as characteristic:

Osteogenesis imperfecta: Multiple, mostly intraperiosteal fractures, often showing areas of bone resorption at the seat of fracture. Excessive callous formation. Increased permeability of all bones of the body.

Rachitis: Increased permeability of the bones. Zone of ossification is irregular and not sharply defined. Cupping and saw-toothing of distal ends of epiphyses. Uneven intensity of the shadow at the ends of the diaphysis. Thickening of the corticalis on the concave side of the bone. Deformities.

Scorbutus: An irregularly circumscribed shadow of varying diameter and intensity at the end of the diaphysis at the seat of new bone formation. This shadow enables one to make an early diagnosis of scorbutus when all other classical signs of scorbutus are absent, provided it is possible to exclude congenital syphilis. This shadow is transverse and located in the youngest diaphyseal zone. Found most frequently in lower extremities. Subperiosteal and intermuscular hematomata, when demonstrable, are of great value for diagnosis. In cases in which separation of epiphysis occurs the characteristic transverse shadow disappears.

Tuberculosis: A primarily destructive process without evidence of a tendency to bone stimulation. Increased permeability of the bones of the region affected.

Osteomyelitis: Demarcation of the sequestrum and unusually early and extensive ossificatory activity of the inflamed periosteum.

Syphilis: In the first weeks of life the osteochondritis syphilitica appearing as either a homogeneous transverse shadow or a streak interrupted by lighter transverse streaks. The epiphyseal line being outlined toward the diaphysis and toward the epiphysis as a serrated ribbon-like shadow. The younger the infant, the more marked is the osteochondritis. In later syphilitic lesions the periostitis ossificans is characteristic, giving the bones the appearance of a double contour.

Basing our conclusions on the above considerations, we feel fully justified in regarding the case presented here as osteogenesis imperfecta tarda with secondary rachitic changes.

CLINIC OF DR. MILTON M. PORTIS

COOK COUNTY HOSPITAL

AN UNUSUAL CASE OF CARCINOMATOUS METASTASES IN BONES SECONDARY TO CARCINOMA OF THE STOMACH

April 22, 1919.

THE first patient I wish to present is a man forty-six years old, a Lithuanian laborer, and married. He was admitted to the hospital March 6, 1919.

Present Complaint.—Pain in the left side; shortness of breath; occasional bloody sputum; weakness and loss of weight.

Onset and Course.—Eighteen months ago, September 21, 1917, the patient's feet, scrotum, abdomen, and face became swollen, and the doctor said he had kidney trouble. He was under the care of a doctor for seven weeks, and was then all right up to May, 1918, when he started to cough and had to sit up a great deal. He kept on working until July, 1918, but had to quit on account of shortness of breath and weakness. He has gradually been losing strength since then, and finally could not walk alone at all, and went to bed about Christmas, 1918. The shortness of breath has been present continuously since July, 1918, and is about the same now. Whenever the patient walks about or exerts himself he gets very short of breath.

Hemoptysis first appeared in July, 1918, and is still present. He coughs up a bloody sputum from one to three times a week. At times he coughs up small pieces of material that the patient states looks like lung tissue to him.

Pain.—The pain is of a sharp, stabbing character, and has been present since Christmas, and is present every time he coughs. He has had a vague lumbar pain for months, but this does not cause much trouble.

Chills and Fever.—These symptoms were very frequent, especially so for the month before Christmas and the month following. He had a temperature ranging from 102° to 103° F.

Night-sweats were present during September, October, and November, 1918. They are not present now.

Weakness has been gradually becoming more pronounced since May, 1918, until now the patient can hardly walk.

Loss of Weight.—He has lost 30 pounds since August, 1918.

Cough appeared in April, 1918, but he had been coughing some for the last four years. He has been a coal miner for the last eighteen years.

Family History.—His wife is living, but has some lung trouble also. All the children are well. No friends or relatives have had lung trouble so far as is known. His wife has had five miscarriages, one still-birth.

Past Illnesses.—Medical: Kidney trouble eighteen months ago. Surgical: Has had no operation. Venereal: Denies all venereal infection.

Physical examination reveals a dyspneic white male aged forty-six, who is in a very much weakened condition. The temperature is 98° F.; pulse, 92; respirations, 24; blood-pressure: systolic, 118, diastolic, 68.

Head.—Negative.

Eyes.—React promptly to light and accommodation.

Mouth.—Teeth are worn and carious; pyorrhea is marked. The tonsils are small and submerged.

Chest.—Expansion is equally great on both sides; right apex is dull and the breathing over this area is bronchovesicular. Numerous moist râles are heard all over the chest.

Cardiovascular.—The pulse is rapid and small. There is no evidence of sclerosis in the peripheral vessels. The heart borders are normal and there are no adventitious sounds.

Abdomen.—Normal in contour. A moderate amount of tenderness is present in the epigastrium. No mass can be felt. The liver and spleen are normal.

Genitalia.—Negative. The prostate is not enlarged.

Reflexes.—Show no abnormal findings.

Extremities.—The right hip is tender and motion is limited. The patient experiences a great amount of pain on motion of right hip. There is definite tenderness over the lumbar spine.

The urine, on repeated examination, did not show any abnormal finding. The red-blood cells were 4,000,000; the white



Fig. 15.

blood-cells 12,000, and the hemoglobin 76 per cent. The blood Wassermann test is negative.

I wish to emphasize that in moving his right side he complains of pain in the hip. There was a sense of grating in the hip. He complained of tenderness on palpating his back in the lumbar region. He was sent to the x-ray room and there we discovered

definite evidences of trouble in the bones. In this plate (Fig. 15) you will find that the acetabulum is about gone. In this plate (Fig. 16) you will see the outline of the second lumbar vertebra on the left side shows a definite deformity. The acetabulum, if you look at it closely, shows a great deal of bone destruction.

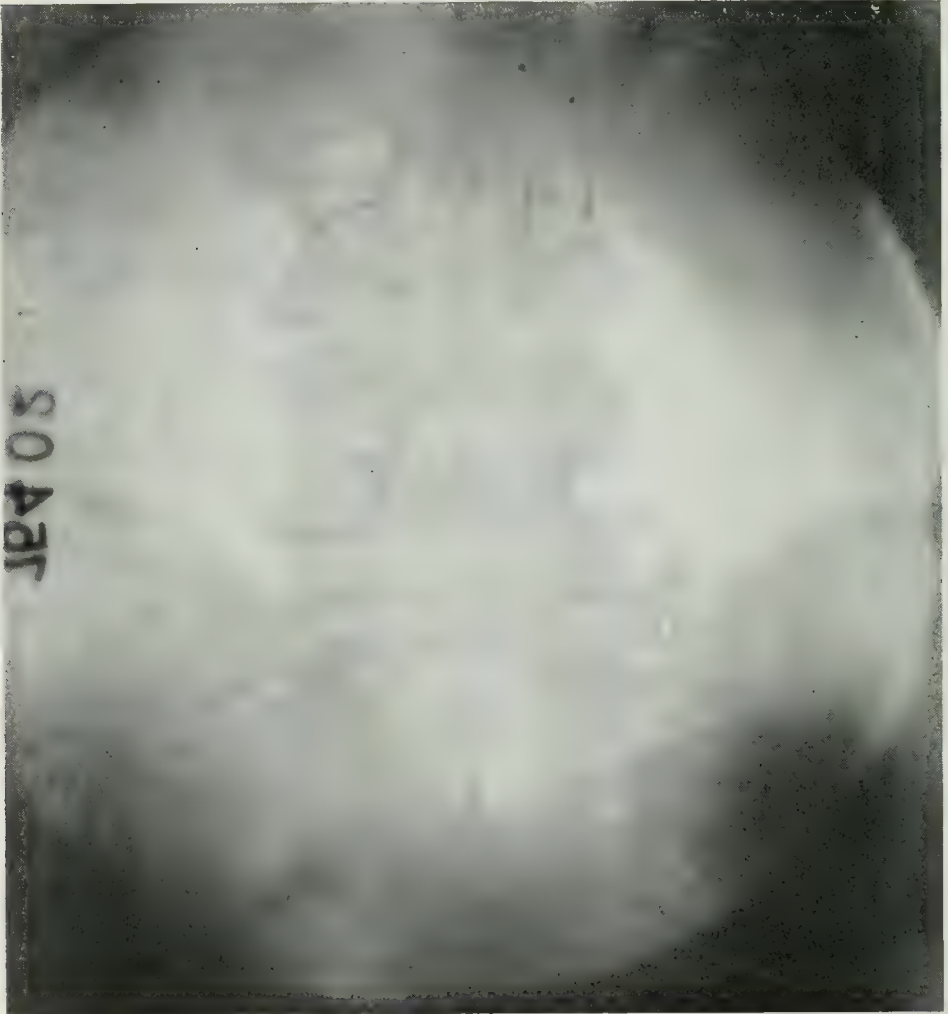


Fig. 16.

One of the other plates (Fig. 17) shows that the rib is destroyed in its inner third. It comes to a termination and there is no more rib tissue; the rib on the other side is perfect in its outline. Dr. Blaine suggested that the patient probably had a metastatic process in his bones.

Although I had thought the case one with renal or pulmon-

ary pathology, the *x*-ray evidence suggested that we were dealing with a malignancy with bone metastases.

Where could the primary tumor be which would give rise to secondary tumors in the bones? The common thing to think of would be the prostate. *x*-Ray men have emphasized for some years that the finding of metastatic growths in the bones indicated carcinoma of the prostate, and that is commonly true.

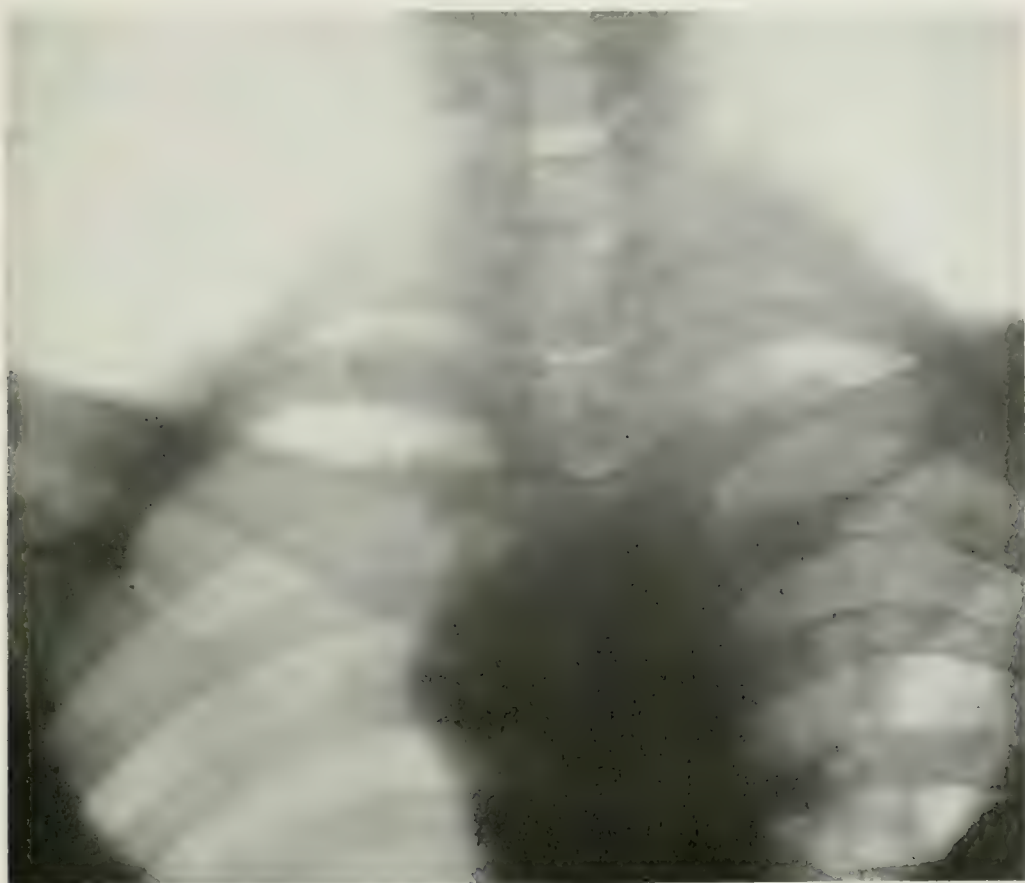


Fig. 17.

The man had a cystoscopic examination and did not show a pathologic change in the prostate.

To further complicate the situation, the stomach test in this case showed abnormal findings. There was no free hydrochloric acid, a total acidity of 38, and blood was present. A bismuth examination of the stomach was made by Dr. Blaine, who reported a deformity at the pyloric outlet which does not include the entire pylorus. The defect is irregular and the *x*-ray

interpretation of it was that it was probably malignant. In addition, there was a small six-hour residue and reverse peristalsis was seen to originate at the lower end.

Without any history or complaint from the patient of trouble in the gastro-intestinal tract a careful investigation reveals pathologic, laboratory, and *x*-ray findings which speak for a probable malignancy of the stomach. I consider this to be a case of latent carcinoma of the stomach with metastatic growths in the bone.

If that is true, it is a very unusual case, for bone metastases from stomach carcinoma are very rare, only 2 per cent., whereas, on the other hand, such changes are very common in prostatic carcinoma.

I emphasize this again, for every now and then you will run across a patient who has a stomach carcinoma, and it will be found on *x*-ray examination or operation or at autopsy. There will be nothing in the clinical story of the patient that will suggest to you a stomach disorder. You will see him for various complaints, and it may not occur to you that the primary trouble is in the stomach. Occasionally metastatic growths will be found in the culdesac, and give evidence of a rectal tumor, which is not a primary rectal tumor at all. On investigating it more closely you will find that you have a carcinoma above, and by gravity the cells of infected material, whatever it may be, have gone downward and set up a growth there, and the patient will complain of rectal disturbance. You have to be on the alert at all times before deciding whether a tumor is primary or secondary, and if it is secondary always investigate the stomach as one of the sources of trouble.

ACUTE PYELITIS SIMULATING INTESTINAL OBSTRUCTION

DR. PORTIS: Several of you gentlemen have examined this young man. I wish you would tell me what you think about him. What was your diagnosis?

VISITOR: It was not easy to come to a conclusion. The examination did not tell me very much.

DR. PORTIS: Who else examined him?

SECOND VISITOR: I examined him, but did not decide what the trouble was. I thought of an acute gastritis on top of a chronic one, and also of a cholecystitis because of the pain and its location. The pain was more like that of a kidney condition.

DR. PORTIS: What did you think?

THIRD VISITOR: I thought it was a kidney affair because he had only one attack until about two weeks ago, and the symptoms seemed to fit into that better than anything else.

DR. PORTIS: This man came to the hospital on the 9th of April, 1919. He is twenty-five years old, an American, single, a soldier, and the examining room diagnosis was ptomain-poisoning. He complained of pain in the abdomen and vomiting.

Onset and Course.—Last evening at 6 o'clock the patient stated that he ate roast veal, bread, cooked tomatoes, potatoes, and coffee in a restaurant for dinner. Half an hour later he was seized with a severe cramp in the upper left portion of the abdomen and had a desire to vomit at this time. The pain in the abdomen, you will notice, began one-half hour after the patient ate his dinner. It came on suddenly and gradually grew worse and worse. The pain is paroxysmal, coming on as if the patient had cramps. Vomiting first occurred at about 10.30 P. M., and since that time the patient has vomited twice.

Past History.—Medical: No history of stomach trouble. No urinary disturbance. Acute indigestion five years ago. Malaria five years ago. Has been troubled with gas from the stomach after each meal. Venereal history: Chancre is denied;

gonorrhoea eight years ago. Surgical: He has had no operations. Alcoholism is denied.

General History.—He has had no previous attacks of similar nature. The bowels have been all right and he has been perfectly well for the last week. He has occasionally felt a heavy sensation in his stomach after eating. He has passed blood with the stool at times for two years.

The cramps and pain have not stopped since he was admitted. No results were obtained from cathartics or enemas. He came in with the picture of an acute gastric affair with a great deal of pain, some nausea, and a little vomiting. He was in great distress when I saw him shortly after admission. The pain was epigastric and localized in the left upper portion of the abdomen, and this region was distinctly rigid as compared with the right side. On admission his temperature was 98.6° F., pulse 56, and respiration 24. The temperature rose to 99.4° F., but the pulse remained slow. The next morning the temperature was 100° F., pulse only 70; the next day temperature was 100.2° F., pulse 76. He had a normal temperature in the morning and a slight afternoon rise until the last two days, when the temperature has been normal both morning and afternoon.

When I examined the patient he first looked as though he was in an acute surgical condition. There was rigidity of the upper left quadrant and an anxious look; he was nauseated and had been vomiting a little. No results were obtained from an enema, and the question arose of the possibility of an intestinal obstruction. Shortly after that he passed some blood. Whether this was due to trauma from the colon tube or from something that bled by itself, we could not definitely decide, but he had a history of having passed blood before coming to the hospital. That brought up the question as to whether or not we had a neoplasm in the colon which was causing an acute intestinal obstruction. On further examination within a few hours the patient showed distinct tenderness on the back in the left side. When suddenly struck on the back in the lumbar region in the right side he would move slightly, but he would almost jump out of bed when he was hit with the same force on the left side. That is a test that is

made for determining an acute renal or perirenal trouble. Urine which was obtained at first showed a small amount of blood. The urine since has shown pus and blood constantly. There was no sugar. Casts were not found at any time. The leukocyte count was 22,000.

Because the evidence was not clear for kidney or for bowel trouble, it was decided that before he has a laparotomy we would cystoscope him and catheterize his kidneys, if his condition did not become worse in the meantime. He was catheterized and a cloudy urine was obtained from the left kidney. The cystoscopic report states that the left ureteral mouth was swollen and red; urine from this side was smoky, due to blood. Urine from the right side was slightly cloudy. Both catheterized specimens were cultured. Cultures from both ureters reveal a Gram-negative bacillus identified as a member of the colon group. Special stains for tuberculosis were negative. This is some of the urine this man is passing now (exhibiting specimen). That material in the bottom of the bottle is pus. There are many organisms present in the urine now which were not present in the original catheterized specimen.

Since the patient has been catheterized and measures have been instituted for quieting down his bowel condition he has become more comfortable and, of course, has not been bleeding. I have asked for an *x*-ray examination of the kidney to see if it would show anything, and on the left side there are numerous shadows which correspond with the shadow of the kidney, and these look like multiple calculi in the left kidney (Fig. 18). That is an old trouble which preceded the present condition.

The sigmoidoscopic examination revealed a polyp hanging from the posterior wall of the rectum about 2 inches inside of the sphincter. Its tip was ulcerated and bled easily.

I feel that this young man has had calculi in his kidney for some time without having the ordinary manifestations of renal colic. This present attack is due to an acute pyelitis caused by the colon bacillus. Dr. Carl Meyer, assistant medical warden, tells me that he is called upon almost every week to differentiate between a surgical abdomen and a pyelitis, espe-

cially in children. These cases of pyelitis come up so suddenly and produce such a marked picture and look so much like an abdominal affair that they are very confusing. It was only by studying this case closely that we were able to differentiate it at all. With the history of blood in the stool, the distinct rigidity of the left side, the marked tenderness in the abdomen on that

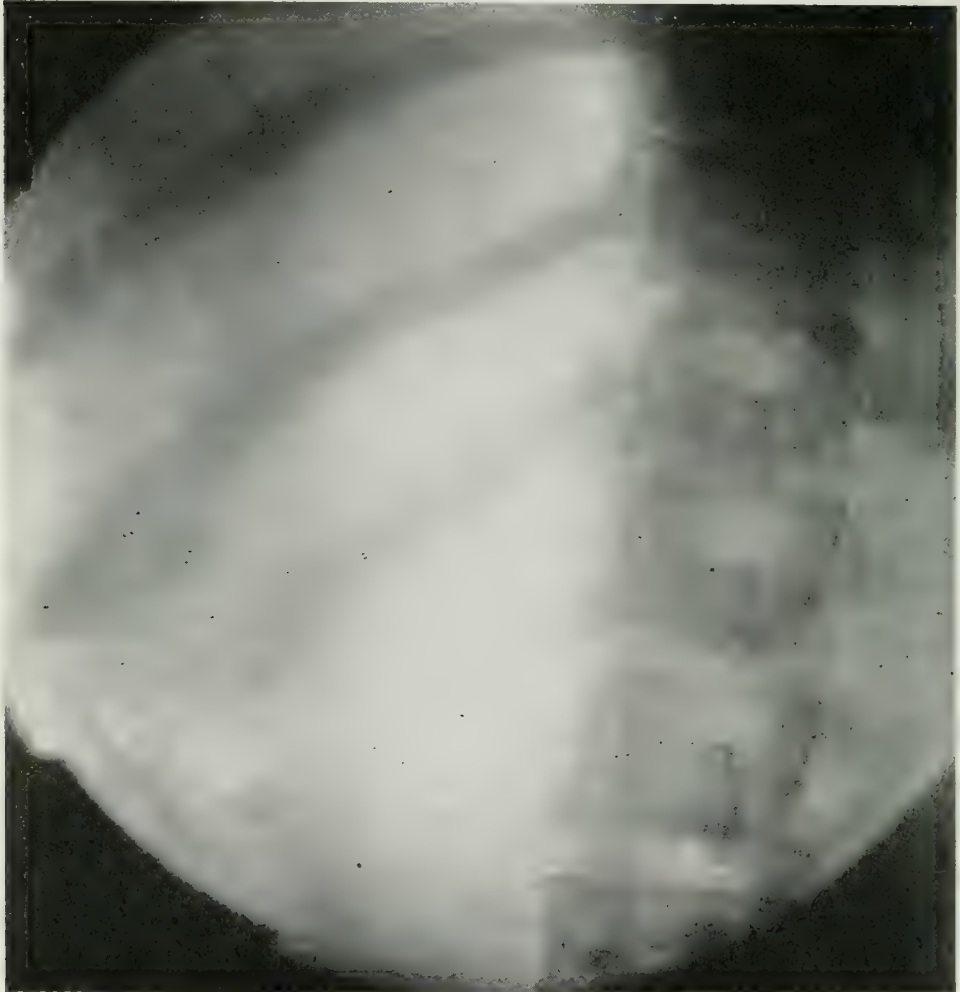


Fig. 18.

side, the colon required careful investigation. The patient has quieted down and is getting along very nicely, and now we are studying the conditions he had before this acute condition set in. We are trying to interpret the kidney shadows, to determine whether they are renal calculi or if they represent some tuberculous trouble, or some old trouble which has left little cavities or calcareous deposits on the left side.

CARCINOMA OF THE ESOPHAGUS TREATED WITH RADIUM

A LABORER, a Lithuanian, thirty-seven years of age.

Present Complaint.—Inability to swallow.

Onset and Course.—The patient states that fourteen months ago he developed a severe cold and hacking cough and had difficulty in swallowing solids. Liquids did not give any trouble. This lasted seven days and then the trouble disappeared. Two months later the same trouble recurred and lasted a few days. He has had intermittent trouble lasting a few days for the past two months, and it has been growing progressively worse. For the past two weeks even fluids give him trouble. On former occasions he experienced sudden relief when he would succeed in eructating a large amount of food and gas. For the last week he has been unable to swallow anything at all and he has a constant substernal distress. He has lost a great deal of weight and feels very weak.

Previous History.—Aside from pneumonia, which he had twelve years ago, he has had no illness of any consequence.

Venereal History.—Denies all venereal infection.

Family History.—No members of the family have had anything like his present trouble. There is no history of tuberculosis or cancer in the family.

Habits.—He has been a constant moderate drinker for many years.

Physical examination reveals a poorly nourished male of good development. There are no eye muscle changes. His eyes react promptly to light and accommodation, and the nervous system does not show any abnormalities. There are no glands to be palpated in the anterior or posterior cervical region. He has small inguinal glands; there are no epitrochlear glands to be felt.

The chest is barrel shaped and his lungs are emphysematous. There is a definite substernal dulness. There is no tracheal tug.



Fig. 19.

The heart is normal in size, the tones are clear, and the blood-pressure measures systolic 130, diastolic 90.

The abdomen reveals no abnormal findings. The liver and spleen are normal.

The extremities are negative.

The blood shows a distinct secondary anemia of moderate degree. The blood Wassermann is negative.

The urine reveals nothing abnormal.

On passing a stomach-tube an obstruction was met at a distance of 15 inches from the incisor teeth. On withdrawing the tube there was a small amount of blood in the opening at the end.

Dr. Blaine made an *x*-ray examination and reported a complete obstruction of the esophagus just above the junction of the middle and lower thirds. The ragged character of the outline at the seat of obstruction indicated a malignant disease (Fig. 19).

With considerable difficulty the patient finally succeeded in swallowing a silk thread, and when it was down into the bowel and permitted tension a bougie was threaded on the silk thread and the stricture was dilated. Two definite obstructions were met with, separated by several inches. On withdrawing the bougie a small piece of tissue was found adherent to it, and it was sent to the laboratory for examination. Dr. Stangl reported it to be tissue from a squamous-cell carcinoma. After the dilatation the patient was able to swallow very well for several days, when the trouble recurred. It was then decided to give him radiotherapy, and Dr. Hanford was kind enough to carry this out. Using the silk thread as a guide, a capsule containing 60 milligrams of radium was introduced into the stricture and left there for an hour and a half. This was repeated at intervals of a few days. The pain rapidly grew less, and although for the first three days the obstruction was more marked, probably due to edema resulting from the treatment, yet within a few days the patient was able to swallow, and has been able to take nourishment without any difficulty since that time.

CLINIC OF DR. RALPH C. HAMILL

WESLEY HOSPITAL

A CASE OF CEREBRAL LUES TO BE DIFFERENTIATED FROM ENCEPHALITIS LETHARGICA

May 12, 1919.

BECAUSE this case was seen during the prevalence of the after-effects, or sequelæ, of the epidemic of influenza it was seriously considered as belonging among these sequelæ; hence the importance of the diagnosis.

The case was seen with Dr. Osgood. A portion of the history as taken by the intern and subsequently by myself is interesting because of the two points of view from which it was written. Many injury cases are handled in this hospital, and the resident men are accustomed to consider each case from the standpoint of a possible injury. The intern's history is as follows:

Five weeks ago, the end of the first week in January, the patient was struck on the bridge of the nose by a ladder (the force of the blow is entirely conjectural inasmuch as it is not stated whether the ladder fell or whether it just walked up and hit him). This injury did not seem to be severe, no bones were broken, and the skin was not even discolored; only a slight swelling was noticed. The patient noticed no ill-effects from the blow until two days later, when severe pains set in over the eyes while he was working. These pains became progressively worse and passed around in front of the left ear, then posteriorly to the mastoid region, then upward to the vertex and down on the right side, about right ear to right eye. About this time the eyes became bloodshot and the face would swell up and be purplish in color. The above pains have continued to the present time (February 10, 1919).

One week ago yesterday (Wednesday) the patient became delirious and had his first convulsion at 6 in the morning. This lasted for an hour, during which time the patient shook all over and had great difficulty in breathing. At noon of the same day he had another convulsion which lasted for an hour and a half. Between the two convulsions he was rational and felt better. He continued to have convulsions daily. After three or four days the condition was so bad that even between convulsions he shook all over, the mouth twitched constantly, and the eyes had a glassy look. The patient complained of being unable to see clearly and also of seeing double. He talked constantly and incoherently, but seemed to understand what was said to him even during a convulsion. There was a marked tender spot in front of the left ear which was rather constantly present. The convulsions continued at intervals of about eight hours until the patient entered the hospital.

Comment.—The striking thing about this history is the emphasis on the apparent causal relation between the injury and the convulsions. The history as I obtained it was, in short, as follows:

In November, 1918, the patient had the grip, headaches, backaches, weakness, no gastro-intestinal disturbance. He was in bed for ten days and the remainder of three weeks he stayed around home. He worked for ten days, during which time he felt all right except that he was weak. Then early in January he was struck on the nose such a blow as he would have paid little attention to had he not subsequently developed pain in the head. He was not unconscious and worked the remainder of the day—seven hours. These pains, as described in the previous history, continued up to the time of the convulsions.

This history is written with the causal influence of the infection upon the convulsions emphasized beyond that of the blow. In the light of the subsequent developments my mistake was almost, if not quite, as great as that of the intern.

The patient's previous history contained the fact that he had had acute suffering in the left ear eighteen years ago, which subsided only after the ear-drum had been lanced. This had

returned one year ago, the abscess had finally broken, and he was relieved. He has had headaches more or less frequently during the past twelve years.

The family history is negative. He has three children living and well; the oldest is nineteen, the youngest fourteen. Habits moderate. No definite venereal history.

Examination, February 13, 1919.—The patient was rather stupid and showed a tendency to convulsive twitchings of the face as he talked. The deep reflexes were all brisk, the abdominals were diminished to a point where it was questionable whether they were present or not. The cremasterics were diminished and equal. When the foot sole was stroked lightly the great toe had a tendency toward dorsal flexion. When medium pressure was used, plantar flexion was the rule. The right pupil larger than the left; neither reacted to light; it was impossible to get him to accommodate. There seemed to be slight facial paresis on the right side. There was distinct rigidity of the neck with a slight Kernig sign. There were no distinct sensory changes demonstrable.

The spinal fluid came out under very much diminished pressure; there were 170 cells to the cubic millimeter, mostly lymphocytes. The Nonne reaction was positive, and with nitric acid there seemed to be a marked increase in the amount of serum albumin. The Wassermann on the blood and spinal fluid was declared negative.

The drum-heads showed nothing abnormal. Dr. J. Gordon Wilson found no disturbance of labyrinthine reactions.

During the patient's eight weeks' sojourn in the hospital the temperature, at first slightly elevated, usually approximating 100° F. rectal, showed considerable variations; during the latter portion of his stay daily variations from 98° to 100° F. being noted. His pulse throughout was rapid, usually over 100, until the last week of his stay.

On February 24th the facial paralysis became well marked both in the upper and lower divisions. The right pupil was a little smaller than the left; both were dilated; both reacted slightly to convergence and the right slightly to light. Attempts to

look to the left showed fine nystagmoid twitching, with some inability to follow the finger to the extreme lateral positions. On the next day the pupils were practically equal. Three or four days later the right pupil was noted as larger than the left.

A lumbar puncture on the 20th of March showed 15 cells, all lymphocytes, to the cubic millimeter, the Nonne was positive, and there seemed to be a marked increase in the pressure from the previous puncture.

The Wassermann reaction on both the blood and the spinal fluid was declared negative throughout. Urinalysis showed a moderate amount of albumin upon two different occasions. This practically disappeared after the first week.

During the first week three white blood counts were made, averaging 11,500. Two weeks after entrance the count was 13,000, 27 per cent. being lymphocytes.

In spite of the negative Wassermann reaction and of the failure to obtain venereal history, I was inclined to consider the case one of cerebrospinal syphilis, largely because of the character of the convulsions. There is something that is perhaps not pathognomonic, yet highly characteristic, of syphilitic convulsions in contradistinction to the convulsions of trauma, brain tumor, and poisoning. One sees convulsive conditions in paresis and to a less degree in cerebrospinal lues, in both of these conditions due to vascular disease rather than to the presence of gummatous new growth, during which there is but a relatively small clouding of consciousness. Also, between the major convulsive seizures there is an intermittent to almost constant twitching of some muscle group or other. It was this combination of retained consciousness and facial twitching between the major attacks that led me to consider lues rather than trauma as the cause.

The condition of the pupils suggested cerebrospinal lues. The right was distinctly ovoid in outline and almost fixed. The left, under observation, became larger than the right, and then smaller than the right; it was more regular in outline.

The number of cells in the spinal fluid agreed very well with what we find in cerebrospinal lues. The slight elevation of

temperature also fitted quite well into the picture of cerebrospinal lues.

Because of these facts the patient was given intravenous injections of neoarsenobenzol.

During the period of his stay in the hospital he had one setback on the 23d of March. Examination at that time showed that attempts to look to the left developed fine nystagmoid jerkings, and there was some inability to follow a finger with the right eye. During this setback he had severe headache and dizziness.

On April 3d he left the hospital much improved. The pupils still were uneven, neither reacted well to light, though there was a very slight movement, especially in the left. Both reacted slightly to convergence. The pupils had become much smaller, contracting from a moderate dilatation to a rather well-marked contraction.

On May 1st he returned to the hospital again, saying that he had no pain, but was almost always a little dizzy and tired easily. Examination at this time gave the following findings: The plantar reflexes were not frankly normal; in fact, the right great toe moved very little. There was slight fanning in response to the Chaddock test. The deep reflexes were all increased, the abdominals slightly diminished. Both pupils reacted to accommodation; the right reacted very slightly to light.

Lumbar puncture disclosed 15 lymphocytes to the cubic millimeter, the Nonne was positive, Wassermann positive with 0.6 of a cubic millimeter of spinal fluid. The Lange showed the low curve of cerebrospinal lues.

Comment.—In ordinary times, in spite of the negative Wassermann, I would have had very slight hesitancy in considering this case a frank expression of luetic involvement of the central nervous system. The character of the convulsions, the pupillary changes, the facial paresis, the transient diplopia, the cellular and globulin content of the spinal fluid, all taken together made a case sufficiently strong to warrant one's paying little attention to negative findings so far as the Wassermann reaction was concerned. This attitude of mind, fortunately

in this case was borne out by the positive reaction obtained on the last fluid, examined on May 1st. There are, however, so many cases of acute involvement of the central nervous system coming on either because of or following influenza that one hesitates in ruling out such a complication, especially when the laboratory returns a negative Wassermann report.

I think we may safely neglect altogether the trauma as a determining factor. However, it may play a rôle in the establishment of a *locus minoris resistentiæ*. The blow on the head may have caused the infection, or the infectious agent, to localize in the brain. The rôle of trauma as the causative factor of paresis and locomotor ataxia has long been a subject of controversy. At one time it was even considered that certain cases of disease of the central nervous system which we now feel certain to be due to syphilis were caused by trauma. Many awards for personal damages have been granted because of the outbreak of parietic or tabetic symptoms coming immediately after trauma. It is much more probable that because of the paresis or the tabes the injured ones were careless or clumsy and so came by their injury.

It would be unwise to close the discussion of this case without saying that it is perfectly possible that we have here a man with syphilis whose central nervous system was invaded by some other disease-producing microbe, such as, for instance, produces encephalitis lethargica. There is no way of being absolutely sure that this latter invasion did not occur. However, we must go ahead with the treatment of the patient as a case of syphilis until we can satisfy ourselves absolutely that the negative Wassermann really expresses the absence of the spirochete.

A factor which suggests encephalitis lethargica is the course of this man's disease. The convulsions, the difficulty with vision, the pupillary abnormality, these things found on February 13th, and then after a period of a week to ten days a facial paralysis developing. Such a course has frequently been described in encephalitis lethargica: evidence of involvement of certain centers, a period of days elapsing, and then manifestations of involvement of other centers.

Note.—The man was seen on May 20th, when the pupillary reflexes were almost exactly as has been described, light practically absent, accommodation faint. The other objective findings have disappeared. No mental abnormality has been observed except that the patient's wife has said that he seems to have frequent nightmares in which he talks or screams with terror. He complains that he still feels dopey, tires easily, and any sudden movement occasions dizziness. Another lumbar puncture and blood test are to be made in the near future; in the meantime the antisyphilitic treatment is being continued.

NEUROLOGIC FINDINGS IN A CASE OF ETHMOIDITIS

May, 1919.

THIS well-built young man, twenty years of age, is a type-setter by occupation. Twelve years ago he began to have headaches. They were usually present on waking. They might occur as frequently as two or three a week, or he might go a month or two without them. The pain is described as pounding or bursting. It frequently is very much worse when he lies flat on his back than when he sits up. During all these years he says that the pain has invariably started from the left eye, running back through the left side of the head to the occiput.

Early in March, 1919, two months ago, he first complained of nausea with the headaches and quite recently he has begun to vomit with the headaches. For six weeks he has had dizzy spells, with and without headaches. During the spells he feels as if he were falling to the left. He knows of nothing that starts the headache. It usually lasts all day. The pain has occasionally wakened him at night, but does not usually prevent his going to sleep.

Double Vision.—Two months ago, for the first time, the patient noticed that when he looked to the left he saw double. This lasted for two weeks, during which time the headaches were particularly severe. Then for two weeks he had no difficulty with vision. A month ago the double vision returned and has persisted up to the present time.

Numbness.—On the outer surface and sole of the right foot there is a slight numbness and tingling. This has been noticed off and on for about a year.

Examination revealed a slightly positive Romberg sign. He was unable to stand on either foot with eyes closed. In the finger-to-nose test there was some uncertainty in the right arm. Superficial reflexes were normal. Deep reflexes were everywhere present, the right ankle, -knee-, and wrist-jerks were slightly more active than the left. The plantar reflexes were normal.

He declared that the prick of a pin was not quite as sharply felt on the outer surface of the right foot as on the left. The sensation was described as more of a numb feeling. Joint and tendon sense showed a diminution in the appreciation of passive movements in the great toe on either side. At times this seemed more marked on the right side than on the left. This is graphically illustrated in the following manner: The great toe is grasped firmly on either side of the terminal phalanx and moved through an arc of about 10 degrees upward and downward, and the patient is instructed to report as soon as he feels the motion. The effort is made to move the joint equally with each change of position. This method is a rough imitation of that which was used by Head in his work on sensation. This table is an example of the method of scoring (demonstrating on the blackboard). The Arabic numerals represent correct replies. The x's represent mistakes, *i. e.*, he has said "up" when the motion was down. The zeros represent failures to appreciate change of position.

TABLE						
Right great toe:	Up	1xx	X2X	1X	020	4/10
	Down	1	xxo	10	0X3	3/10
						7/20
Right great toe:	Up	X234	X	1X3	X2	6/10
	Down	123	12X12	12		9/10
						15/20
Left great toe:	Up	1	1XXX5	123	1	7/10
	Down	12	123	1X	123	9/10
						16/20
Left great toe:	Up	1	12	10X4	123	8/10
	Down	12	123	02	1X3	8/10
						16/20

From these tables it would seem as though he makes a few more errors on the right side than he does on the left. However, the difference is not such as would lead one to place any great confidence in these results. One must make allowance for mistakes due to a deficiency of attention and mental alertness.

There is no difference in strength on the two sides. In pro-

truding the tongue there is a slight difference in the height of the arch formed by the drawn-back upper lip, the left side is a little lower than the right.

The plantar reflexes are normal. Gordon, Oppenheim, and Chaddock are absent. The ankle-jerks are brisk, as are the knee-jerks, both of which, as well as the wrist-jerks, seem to be slightly unequal, the right a trifle more brisk than the left. Biceps and triceps jerks are brisk and equal. The cremasterics and abdominals are equal and normal. The pupils react to light and accommodation. The veins of the optic nerve appear slightly congested. The thyroid is rather full. There is no lead line, no signs of anemia, and no hypersensitiveness of the palpable nerve trunks.

The spinal fluid, under normal pressure, contains 3 lymphocytes to the cubic millimeter and there is a positive globulin test.

The important neurologic symptom is the paralysis of outward movement of the left eye. It can be swung from the internal canthus only as far as the middle line. Attempts to follow the finger to the left merely cause nystagmoid jerkings.

Dr. Bookwalter examined the patient two days after admission, finding a purulent rhinitis and sinusitis on both sides, and infected tonsils.

On May 9th it was noted that the left eye moved well beyond the middle line. On the 12th he reported that he felt better, that his last severe headache occurred four days before, vomiting the next day during the persistence of the headache. The left eye could be rotated externally almost to the outer canthus. The pupils reacted to light and convergence, but showed a hippus, an alternating dilatation and contraction of rather slow tempo, each motion occurring about once a second.

There was equal strength in the two hands and he stood equally well on either foot. The handwriting showed nothing abnormal. There was no demonstrable ataxia in the finger-to-nose or finger-to-finger test. There was no past pointing or adiodococinesis. There was some diminution of appreciation of passive movement in the toes, but it seemed equal on the two

sides. Other sensory phenomena were absent. The deep reflexes were brisk and equal.

Three white blood counts made in a period of four days show 12,800, 14,000, and 15,000. The neutrophils run from 68 to 80 per cent.

Comment.—The diagnosis in this case rests between vascular disease in the pons or medulla; new growths in this same region, ophthalmic migraine, and disease in the orbit. Cases of ophthalmic migraine, as far as my experience is concerned, are becoming more and more infrequent. "Ophthalmic migraine" is a term applied to sick headaches with which are associated transient paralyses of ocular muscles. Many cases formerly called by this symptomatic name have been proved to be syphilitic, since the use of the Wassermann on the blood and spinal fluid, especially the latter, has become the routine practice in the investigation of this class of cases. No case of sick headaches with paralyses of any nature should be allowed to escape a lumbar puncture.

This particular case does not exactly correspond to the typical picture of ophthalmic migraine, in that there have been severe headaches for many years and only in this last few weeks have there been the concomitant symptoms of nausea, vomiting, and ocular palsy. Still, I believe that symptomatically this case might be termed one of "ophthalmic migraine." Ophthalmic migraine, as I have said, is a symptomatic name, and it is our task to find the causative condition. In this instance I think we need not hesitate to ascribe the whole disease picture to the findings in the nose. There is a purulent rhinitis and sinusitis, and upon inquiry we find that he has had crusts in his nose for many years; the condition is of long standing, perhaps as long as his headaches.

As signs of the infection we have the nose findings, the infected tonsils, the elevated white blood count with its large percentage of polynuclear cells, and the slightly enlarged thyroid gland.

As evidence that the ocular palsy is dependent upon this infection, we have the fact that rest in bed and nasal spray, re-

lieving the sinus condition, is accompanied by or results in the improvement in function of the paralyzed muscle. Have the nasal or tonsillar infections caused the paralysis of the ocular muscle? is the next question to decide.

The muscles that move the eyeball take their origin from the back of the orbit, where they are separated by only a fraction of an inch from the walls of the ethmoidal cells. Hence, one would be tempted to ascribe the disability in this case to a direct extension of an infection through the wall of the ethmoid, but if such an extension had occurred we would certainly expect some symptoms of involvement of the muscles running along the inner wall of the orbit rather than of the external rectus running as it does along the external wall of the orbit. Also there should be some evidence of disease in the orbit, such as proptosis, or forward bulging of the eyeball; pain both while the eyeball is still and in motion; and evidence of retrobulbar neuritis. The absence of any of these findings should rule against involvement of direct extension. Slight facial weakness, inequality in reflexes, even the numbness in the right foot, may all be considered as indicating that the lesion is in the central nervous system, namely, in the lower part of the pons on the left side at the level of the sixth nucleus.

At this level the sixth nucleus lies about a millimeter below the ependyma lining the fourth ventricle. The fibers running from the seventh nucleus to the exit of the seventh nerve from the pons form a loop around the cells of the sixth nucleus. The fibers of the sixth nerve run almost directly ventralward, that is, to the ventral surface of the pons, in their course passing through the fibers of the fillet and those of the pyramidal tracts.

The fibers of the fillet are conveying upward sensory impressions from the opposite side of the body. Motor impulses are being conveyed downward by the fibers of the pyramid. Over these fibers are also passing impulses from the higher nervous centers, probably the cortex, inhibiting the tonic character of the control exerted by the anterior horn cells over the muscles. If the pyramid is cut, then the spinal centers, the anterior horn cells, are unopposed or are not inhibited by these

higher centers, and we see a condition of increased tonicity with spasticity and exaggerated tendon reflexes. If the fillet is interfered with there are all degrees of disturbance of sensation in the opposite side of the body, from mere subjective numbness, tingling, crawling under the skin, coldness, etc., to complete loss of all forms of sensation.

The objection might be raised that in this particular case symptoms from the fillet and pyramids are too indefinite in comparison to the very marked disability of the sixth nerve. The subjective numbness in the right foot, the disturbance of joint and tendon sense, slightly more marked in the right foot than in the left, and the fact that the reflexes on the right side were a trifle more brisk than those on the left, seem rather inconsequential when compared to the marked change in appearance caused by the paralysis of the external rectus, the high degree of internal strabismus. The fact that the eyeball is maintained in its proper position by a balance between the various muscles and the fact that the muscles turning the eye in are considered as being very much more powerful than those which turn it out, these two facts may account for the apparent inequality in the symptoms. A slight weakening of the impulses passing out over the fibers of the sixth nerve to the external rectus is sufficient to permit of an overaction of the internal rotators of the eyeball, and so produce the internal strabismus.

I have observed two or three times the following phenomena: After complete ophthalmoplegia, in disease of the nuclear centers, with returning function the complete loss of motion of the eyeballs has given way to a paralysis of the external rotators, leaving a condition of bilateral internal strabismus. In one case this internal strabismus disappeared, leaving the eyes in normal balance; in another case the muscles of one eyeball regained complete function, while the internal rotators continued to overwhelm the external in the other. It is such clinical facts as this which lead me to believe that the interference with the sixth nucleus and nerve does not need to be very great to cause the internal strabismus. It is on these grounds that I can reconcile the marked symptoms from the sixth nerve as against

the slight symptoms from the seventh, the slight facial paresis already mentioned, the numbness and apparently unimportant sensory changes in the right foot, and the barely demonstrable difference in reflexes.

The presence of globulin and the few cells in the spinal fluid are evidence of an involvement of the central nervous system, perhaps not strong evidence, but, taken in conjunction with the other findings, they have their own definite importance. In fact, where there are any other findings pointing toward involvement of the central nervous system, I am inclined to put considerable faith in the findings of 3, 4, or 5 lymphocytes to the cubic millimeter of fluid. When I find so few I always make it a point to count at least 5 or 6 different drops, 5 or 6 different films 9 mm. square and $\frac{1}{10}$ mm. thick. When I have found 3 to 5 cells in each such film I feel they are not to be slighted in determining the presence or absence of involvement of the central nervous system.

Ophthalmoplegia or paralysis of the oculomotor nerves is usually symptomatic of some other disease, some infection. It may occur in syphilis both in the early stages and in the late; both as a secondary phenomenon and as a symptom of tabes or paresis. In fact, it is not uncommon to hear of diplopia occurring within the first year or two after the primary chancre in those who later develop tabes and paresis. It has been thought that such early involvement of the cranial nuclei shows a weakness of the central nervous system against the spirochete or a predilection of the spirochete for the central nervous system; hence, it might be considered as a premonitory or warning symptom of the later developing tabes or paresis.

There are no other phenomena that suggest syphilis unless it is that the ethmoidal or other sinus disease that unquestionably is present is of luetic nature. The Wassermann reaction is negative. I think it is perfectly safe to rule out syphilis.

Many cases of ophthalmoplegia were formerly ascribed to cold—a rheumatic neuritis comparable to the typical Bell's palsy of the facial nerve. This, of course, is a peripheral involvement of the nerve. Cases in which cold is the only etio-

logic factor are becoming more and more rare; in other words, more and more of these cases prove to have some definite etiologic factor. Influenza, alcoholism, diphtheria, food poisoning, and diabetes can all cause peripheral or central involvement of cranial nuclei. The fact that the sixth is involved so frequently is, I believe, explainable on the basis I have already mentioned, namely, the necessity for having perfect impulses pass to the external rectus to allow it to hold its own against the more powerful internal rotator.

Sinus disease is, after all, merely a focal infection in a cavity which, because of the swelling of the lining membrane, can become a place where pus is under increased pressure, a place from which septicemic or pyemic (these terms are used in the broadest sense) involvement of the rest of the body may occur.

This young man has had very severe headaches since he was twelve years old. He has had crusts in his nose or discharge from the nose ever since that time. If one observes him carefully a peculiar sunken appearance to the entire nasal structure is apparent. It is not at all the appearance seen in hereditary or acquired syphilis known as the "saddle-back nose," but there is definitely an appearance as if the nasal structure had not developed as fully as the rest of the face. I believe that each time he has had a severe headache it has been because either an ethmoidal cell, a sphenoidal cell, or some other bony cavity tributary to the nasal passages in the head has gotten more or less occluded as the result of inflammatory swelling of the lining mucous membrane. As long as pus is under pressure there is always danger of its invading the lymph- or blood-streams. There is always danger of systemic infection. It is this that has occurred finally in this particular case. The involvement has been slight but definite, and I am certain that if the central nervous system could be microscopically examined at the lower pontine level we would find some of the small blood-vessels running into the substance of the pons surrounding by round-cell infiltration. This is the pathologic background of the phenomena: Sinusitis, probably ethmoidal or sphenoidal, and pontine encephalitis.

The therapeutic measures are clearly indicated. First of all, call in a good nose and throat man; he will provide for the proper outlet for the infected cavities. He will also, in all probability, want to remove the infected tonsils, as they may contribute either to the reinfection of the sinuses or to the encephalitis. The usual measures of hygiene are, of course, not to be neglected.

Note.—Nasal treatment has been instituted, the nose has been sprayed, one of the turbinates has been removed, and the drainage is being re-established. The patient has not had a headache for the past ten days, a rather long period of freedom for him. The nasal treatment was started eight days ago.

A CONSIDERATION OF THE CAUSES OF APPREHENSION

FIVE years ago a professor of neurology and psychiatry gave a talk to a group of physicians on the subject of neurasthenia and hysteria. He drew a definite anatomic picture, differentiating the two conditions on the basis of the neuron theory. That much, I am sure, we all grasped. The elaboration of the idea was too diagrammatic to leave any lasting impression. He attempted to picture the mind as a machine which, if started running at a certain speed, would produce hysteria; at a different speed, neurasthenia. A machine that would grind out mental states as a factory would produce Fords. There was no place in his theory for the interaction of the individual and his environment. According to him the machine ground out its products regardless of the rest of the world. An explanation based on such an idea explains nothing.

Mental states are the result of an instinctive mechanism working upon the materials fed into the machine by the incoming sensory tracts. I say an "instinctive machine"; perhaps it would be more clear if I said a machine whose force and direction of action are determined by certain instinctive ingredients. I cannot define the last save by using the old terms, the instinct of self-preservation and the instinct of self-perpetuation.

Apprehension is a mental state. It is, at least in part, the result of a conflict between the egoism of childhood, an egoism carried into adult life, and the forces of environment. Fundamentally it is a fear of failure; a fear that the egoism, the self-confidence, will not be able to carry through.

To state it a little differently, apprehension is a result of the conflict between instinct and education. Consider for a moment the instinct of self-preservation. It is the urge to live, as truly present in the amœba, the earthworm, and the cat as in the human being. It is merely another way of stating something that we learned in our early studies in physiology: the properties of protoplasm, the powers of assimilation and reproduction, that,

with motion and sensation, are given as the inherent properties of each and every animal cell. That is the protoplasmic expression of the instinct of self-preservation. Cellular life grinds on and probably by its momentum carries life on irrespective of consciousness.

The conscious expression of the instinct of self-preservation is quite another matter. It finds its most common expression in the saying "that makes life worth while." It is an expression of the measure by which we judge each and everything, act, or thought.

In childhood the pleasure-pain formula rules. By that is meant the measure by which all things are judged. The infant likes that which is pleasant, irrespective of further consequences. It will eat all the sweets possible; it reaches for the light, even that of a flame. This standard of measurement is the instinctive one. It is the standard with which we all are born into the world and with which we measure all our early experiences.

Education is the process of teaching that immediate pleasure, or avoidance of pain, is not the measure to use in judging the amount of good in an act or state of mind. Instinct would lead to immediate enjoyment; it is purely selfish, self-centered. The instinctive type of enjoyment is infantile. The enjoyment is taken as though the taker was alone in the world with only his own criticism and consideration to meet. Education has taught us that our acts are performed in an ever-enlarging sphere. The infant knows only his mother and nurse, and has only them to please. The child has his larger sphere of neighborhood and school; the college boy has enlarged his sphere to that of the so-called university. The business man has added to all of these the world of trade, and the statesman acts in the sphere of the world. In each stage the individual has acquired a new and ever-increasing number of critics—others to please. Education is the process of learning that fact and how to accomplish it. Hence, the pleasure of the adult must have to do with something as far removed from self, from instinct, as is possible if he is to meet the demands of his education. Immediate pleasure is instinctive; it is reflex. Its consideration or entrance into judgment is a reflex matter, and, like all other reflexes, such as

knee-jerks, winking, etc., it is more natural and quicker to occur than volitional or thought-out responses. But education has taught us that we cannot live a life on the pleasure-pain basis, that we cannot eat all the candy in sight, that when we see the money lying on the bank teller's shelf we cannot reach out and take it, that because the lady is beautiful we are not justified in going very far with her along the road of pleasure. Hence, we are in the position of entertaining two conflicting motions; we are in the embarrassing position of a chairman refusing to recognize "the previous motion," the sponsor of which is the most powerful man in the community. Our embarrassment comes because we must recognize and give precedence to the motion of education.

To end this rather long-winded dissertation I would put the matter in this manner: We are the seat of a conflict between the forces of instinct and those of education, and our ability to get on in the world depends upon the degree to which we can mold the forces of instinct by education. When we sense impending failure we become apprehensive. It is that state of apprehension that at least, in a very large measure, determines the presence of the neurosis.

I trust I have made it clear that it is my conception that there is a conflict going on, and that our satisfaction in life depends upon the outcome of this conflict, and that if we see danger of losing out we become apprehensive. Now there are many forces which can act on one or on the other side of the conflict. In fact, there is but little that happens that is neutral. Everything that affects our state of well-being makes it more, or less, difficult for us to oppose the instinctive forces. A person with infected teeth or tonsils is absorbing a little poison, has developed a slight anemia, readily tires out, experiences more difficulty in getting satisfaction from the world, is driven, therefore, toward a mental attitude to which the instinctive forces make great appeal; his hold on education is thereby weakened. The man who has sustained an industrial accident is thereby thrown into a frame of mind in which the idea of revenge plays a considerable part—plays much too large a part for him to use that same mind

in a successful manner, and so he reacts more as a child who continues to cry and make itself unhappy long after the time has passed when that crying could punish the person he fancies has offended him.

It is, then, my conception that the various factors forcing an individual to be governed by the infantile attitude toward pleasure are also leading him to fear his hold on education, his ability to satisfy the world. These things, then, are causes of apprehension.

The immediate complaints of an individual suffering from apprehension are, usually, inability to concentrate, difficulties with memory, interference with sleep, poor appetite, palpitation, sense of fullness in the head, cold hands and feet, and because of all these things, nervousness or unhappiness. The first two are purely mental traits. The sufferer says, "I can't control my thoughts." Of course he is afraid of failure; he is also afraid of insanity. The other complaints he fears are signs of weaknesses of one sort or another that will eventually interfere with his ability to get on. "If I can't sleep and can't eat, how can I go on working?" Or, more seriously, he says, "If I can't sleep I'll lose my mind."

It is almost impossible to prove that this failure of concentration is due to the fact that attention is somewhat distracted from the immediate necessities by the conflict that I have been talking about between instinct and education, and yet it is this distraction of attention that allows neurotic symptoms to develop. Such a statement finds its most definite proof in such conditions as the following:

A forty-year-old woman whose husband has died, and who is living at the home of her daughter, is brought into the hospital at 3 A. M. by a police sergeant, not knowing her name, her age, her civil state, residence, or any other details concerning her individuality. It turns out that at 2 A. M. her daughter heard a noise from her mother's room, but, thinking nothing of it, went to sleep again. In the morning the mother had disappeared. The police sergeant says that shortly after 2 A. M. this woman came up to him much in the manner of an amateur

street-walker. It was because of his appreciation of her amateur standing, as well as the fact that he was a guardian of public morals, that led him to bring her to the hospital, when, upon questioning her, instead of appreciating her advances as she had expected, she denied all knowledge of herself. Things had happened just before this experience which made it appear to this forty-year-old grandmother that from now on she would have to earn her own living, and that she would have to depend on the forces of education rather than upon those of instinct for her satisfaction, for her ability to get ahead. She had been obsessed by a conflict between instinctive desires and education. In this particular instance the desires were sexual and she saw no means of legitimately gratifying them. Education was too strong in her to allow her gratification in any but a legitimate manner. Hence, when the instinctive promptings became sufficiently powerful she had to forget education. She had to forget all the things she had learned about herself—her name, her age, place of residence, etc., etc. When she had successfully forgotten or cast off from herself all sense of appreciation of the fact that she lived in the larger sphere of adulthood, she was able to attempt to gratify the instinctive desires in an infantile or direct manner. She was an example of so-called dual personality, an individual who was fortunate enough to be able to forget what she did not wish to remember.

The apprehension of self-criticism, the fear of what others might say of her in case she should offend social convention made her forget all these critics. She forgot who she was, and that included what she was to the rest of the world. This enabled her to act in a manner impossible to one who would retain self-respect and the respect of others—family, friends, etc. She was able to forget because of the intensity of the conflict; because of the amount of distraction from the facts of the present.

Another typical example of the effects of conflict producing a state of apprehension is the following: A very pretty woman of twenty-five living in a small-sized city came to the office complaining that six months before she had begun to be nervous, unable to concentrate, had lost interest in her work, had been

having attacks of palpitation, and could not sleep well. The persistence of these symptoms had made her despondent and depressed. After considerable talk she let slip the fact that she thought men looked at her in a peculiar manner on the street. Because she was pretty, twenty-five, and from a small city I was interested to observe that she wore no engagement ring. I asked her if she was engaged; her eyes filled with tears and she said "No, but I have been." Careful and diplomatic inquiry brought out the following facts: She had been engaged to a boy with whom she had gone for several years. As they were planning soon to be married she allowed things to go further than she had expected, and they had intercourse; this happened several times. Then he developed tuberculosis and died. Fortunately, she had not conceived. The essential and interesting part of the case then developed; it was her attitude of mind, determined by the conflict such as I have spoken of. One might think that the fact that her fiancé had died was sufficient cause for the neurosis. But it is not the external event that causes the serious trouble; it is the individual's reaction to the event. This young, well-educated woman found herself suffering from remorse. She also found that the promptings of instinct, the desire for pleasure, in spite of the remorse, would frequently come and upset her whole mental quiet. This was brought out by inquiring into the cause or quality of the sleeplessness. During the day she had been able to get along fairly well; the distractions of the moment were sufficient to keep her mind occupied. After she had gone to bed, and these distractions no longer could help, her mind would revert to the experiences that she both feared and desired. The instinctive side of her wished them repeated; the educated side condemned the desire. Hence, she was constantly aggravated and disturbed by the conflict of the two lines of thought. She became so obsessed with the necessity of keeping her mind off the tempting thoughts that she was unable to concentrate or get interested in other matters. Perhaps the principal fear that she had was that she was not the sort of a person to get on, was too immoral to be able to keep her place in the world; of course, there was also the fear of insanity.

After a complete exposition of her side of the case and my assuring her of the perfectly natural and logical development of her ideas, she immediately became very much less concerned and much less worried. She had thought about these things so much, had become so critical of herself, that she had begun to see criticism in the faces of others; not only criticism, but a certain amount of understanding—the strange look in the faces of men that she passed on the street. When it was explained to her that of course men looked at her because of her natural physical attractions, and that they really could know nothing of her thoughts, but that she merely fancied that they did, another great load seemed to be lifted from her mind. She had begun to fear that people could read her thoughts.

This brings up the subject of the physician's ability to meet the subject of apprehension. Apprehension is an attribute of the mind of the child. There are so many things that it does not know, so many things constantly happening from unseen causes that an apprehensive state is almost to be expected. A child is being constantly corrected and threatened as a part of its education. Explanation of the possible dangers, and rendering familiar the things which because of their unfamiliarity can frighten are the ways in which we meet apprehension in the mind of the child. Most adults are sufficiently unintelligent about their mental processes to need explanation and reassurance when these mental processes seem to threaten. Hence, if the physician can understand his patients, can work back to fundamental things from the avowed symptoms, can translate attacks of palpitation into terms of temptation and repression, he can make the patient realize that his mental processes are the expected and not the abnormal. If he can do this much he has gone a long way toward the relief of the state of apprehension. Repeated explanations and reassurance will help in many cases where all other medical treatment will simply fall flat.

A Point in Functional Diagnosis.—There is an attitude seen in the patient suffering from functional disorder which when recognized aids greatly in diagnosing the disorder as functional. It expresses itself in the evident desire of the patient to discover

the effect made upon the examiner by the exhibition of the symptom. For example, a woman who was complaining of great abdominal distress was discovered to have an enlarged abdomen simulating in size a five months' pregnancy. The shrewd and inquiring look in her eyes made me realize that she was more concerned with the effect that the symptom had on me than that which it had on her, namely, the pain that she said she was suffering from. Close examination revealed the fact that the diaphragm was being held down and the abdominal wall thrust forward in an attempt to simulate abdominal tumor.

Another example, much more common, is that seen in the functional ataxia. The tabetic with mild ataxia, if he is not neurotic, puts his feet together and shuts his eyes and sways gently to and fro with evident unconcern. The neurotic, on the other hand, as soon as his swaying has been sufficient to demonstrate his instability, pops his eyes open, hoping to see the concern upon the examiner's face that he feels such a grave symptom warrants.

Such an attitude is seen both in the truly apprehensive and in the less truly apprehensive but more consciously controlled traumatic neurotic. In the first instance, in the pure apprehension we have an individual who feels the need of a physical expression of his mental instability or suffering, just as the infant must needs cry. So the neurasthenic and the hysteric is much concerned with the impression that his exhibition of symptoms makes upon the examiner. If this disinterested person is impressed with the physical aspects of the case, the sufferer feels justified in thinking those physical aspects of more importance than the mental, feels justified in directing his attention to the physical, and so helps himself to forget the mental.

In the case of traumatic neurosis the need of impressing the examiner with the severity of the physical symptom, with the degree that such a physical symptom interferes with normal function, is, of course, obvious. I need not emphasize such a need. I would, however, emphasize the examiner's need of keeping his eyes open and constantly observing the attitude of his patient in the expression of each particular sign and symptom.

CLINIC OF DR. CLIFFORD G. GRULEE

PRESBYTERIAN HOSPITAL

PYELOCYSTITIS IN INFANCY

THIS little girl baby, thirteen months of age, entered the hospital with her twin sister on the 30th of April of this year. The complaint of the mother was that the child did not stand, had a weak back, and had lost weight. The history is as follows:

The patient is a twin, born at full term in a normal labor on the 2d of April, 1918. Both twins were breast fed for nearly six months, when the breast nursing was discontinued because of ill health of the mother. During this time the mother states the children were well and no supplemental food was given. When the breast feeding was discontinued Mellin's Food was tried, with poor results, the children vomiting the food. A doctor then advised modified milk, which has been used up to the present time. The child is given a bottle of 8 ounces four times a day of the following mixture: 2 quarts of whole milk, 8 teaspoonfuls of cane-sugar, and 6 tablespoonsfuls of thick oatmeal water. Bread and crackers were given between feedings. On this diet the child has always been constipated, practically every day it has been given castor oil, Castoria; suppositories, or enemata. In September, shortly after changing from the breast to artificial food, the infant had an attack of influenza, but without lung involvement. About the middle of November she had whooping-cough, and in December, measles.

The child is not able to stand; the back is weak. There has been quite marked loss of weight in the last two or three weeks. Head sweating was noticed about six months ago. The extremities were noticed to be weak. In the last week or

so the mother has noticed that the child holds the left leg fixed; this seems tender when moved or touched. The child has never had any convulsions.

The father died four months ago of pneumonia. The first child died at two months of age; three other children, aged eight, six, and four, are living and well. Between the four-year-old child and this baby twins were born, which died at birth.

On examination of the child we find a small, pale infant, whose head appears disproportionately large. The circumference of the head is 46.2 cm. You will notice that the forehead is very high, the frontal and parietal bosses are quite prominent. The anterior fontanel is wide open; the parietal bones do not meet and their edges are soft and pliable. The fontanel is rather tense. I can find no evidence of craniotables. There is no tenderness about the ears nor discharge. The nose is small and the child apparently is a mouth-breather. There is slight difficulty in respiration.

On examination of the mouth we find the child has one upper tooth and three lower. There is no hemorrhage about the gums and the throat and pharynx are negative.

The neck shows no rigidity; there are palpable, both anterior and posterior, cervical glands.

On examination of the chest we find a very definite depression at the lower end of the sternum, with marked rachitic rosary. The sides of the chest at the location of the diaphragmatic attachment are perceptibly retracted at each inspiration.

When the child is sitting you see a marked curvature of the spine.

Going over the lungs we find no râles and no areas of dulness. The heart, too, is within normal limits and is normal both as to rhythm and tone.

The abdomen is quite protuberant; the liver is palpable just below the right costal margin. The spleen cannot be felt, nor can one find any tumor masses.

The extremities show no changes except for the enlargement at the epiphyseal end of the long bones. Previously the legs were held in a flexed position and there was evidently pain on

movement, especially when one attempted to extend them. The musculature is weak and flabby and the skin is loose and without turgor. The epitrochlear glands are not palpable, but we find on the left side a small, palpable intercostal gland.

On entrance this child weighed 13 pounds, 2 ounces. She has now been in the hospital for almost a month, the weight varying as low as 12 pounds, 10 ounces and up to 13 pounds, 1 ounce. During her stay here she has shown no tendency to loose stools; in fact, throughout this time her stools have been normal.

Her temperature has usually varied between 99° and 101° F., but on the day after entrance it rose to 102.6° F., dropping within four hours to 99.8° F. On the 6th of May her temperature went as high as 101.8° F. The temperature has shown a general tendency to come down, but has never been within normal limits.

On entrance the child was given a teaspoonful of cod-liver oil with phosphorus three times a day.

To sum up, we have here a rather thin, pale, definitely rachitic infant, whose physical examination reveals nothing of importance except the rachitic symptoms and deformities. This little girl has been running an irregular temperature ever since the day of entrance, and still continues to do so. The Pirquet reaction done shortly after her entrance into the hospital was negative. Her blood count on entrance showed 2,330,000 reds, 4900 whites, and 75 per cent. hemoglobin. The cerebrospinal fluid showed a cell count of 8, negative Nonne and gold chlorid reaction, and negative Wassermann. I failed to mention in summing up the case the very evident pain on movement of the lower extremities. We have, therefore, to account in this case for two things: first, the temperature, and second, the pain which was present in the lower extremities and the cause of their being held in a flexed position.

Before we take up this case further I want to show you another case, and then we will consider the two together.

This little girl is eleven months old. She is well nourished and appears to be in very good general health. She was operated

upon on the 5th of May by Dr. Fiske. The history previous to that operation was as follows:

The mother stated that the child had been ailing for several days and had been crying a good deal; she acted as though she had an earache, but the mother had noticed no discharge from the ear. She was brought because of pain and tenderness over the right mastoid process. The baby had been a breast baby for about four months and then had been fed on half-milk and half-water with a little sugar. The bottle was given every time the baby cried, which apparently was at least every two hours. How much was given in twenty-four hours the mother could not estimate. The only record of physical examination noted in this case is redness, tenderness, and swelling behind the right ear. There was no discharge from the external ear and the drum membrane was intact. At the operation a curved incision about 2 inches long was made 1 inch posterior to the right ear. The periosteum was divided and soft bone was removed. Some pus was found. The wound was closed and drained with iodoform gauze.

The child had been in the hospital four days previous to the operation, during which time the temperature had varied between 99° and 104.2° F. Following the operation, in spite of the drainage of the mastoid, the temperature still remained high, reaching quite regularly 103° F. I was asked to see this case because of the continued abnormal temperature.

Physical examination of this infant, as you see, reveals practically nothing abnormal except the wound over the right mastoid region, which is practically healed. Otherwise we can find nothing in this case to account for the temperature. A blood count on the 16th of May showed 8000 white cells.

In summing up we have here a child suffering from involvement of the mastoid portion of the temporal bone. Following an operation with drainage the temperature showed slight moderation, but continued abnormally high in spite of the fact that the general symptoms perceptibly ameliorated and the local symptoms largely disappeared.

We have here, then, two little girls about a year old with

irregular fever which we must account for. Let us consider the first case and then go to the second. In this case we can be relatively certain that the cause of the temperature is not a gastro-intestinal disturbance. This is proved by the fact that the child had no diarrhea and that there is no prostration at all. We practically never have a fever due to gastro-intestinal disturbance in which the amount of gastro-intestinal involvement as evidenced by diarrhea and vomiting is not commensurate with the temperature. Nor do we have a fever due to gastro-intestinal disorders which does not show more general involvement than in this case. We must, therefore, look to some disturbance outside of the gastro-intestinal tract to account for this fever. We can at once eliminate the acute contagious diseases, such as measles, scarlet fever, etc., and also such involvements of the nervous system as anterior poliomyelitis and meningitis. There is not and never has been any evidence of otitis media, nor is there present any lung involvement. The throat is clear.

We have now considered practically all the causes of fever in infants which the average practitioner holds before him when he examines an infant. He forgets far too often that it is of paramount importance in these cases of obscure fever to get a specimen of urine. The examination of the urine in this instance shows that it is loaded with pus. I will take up later, in discussing the general subject of the disease, the treatment to be used in these cases.

Let us now consider the second case: Here we have a perfectly normal girl baby who, following operation, continues to have a high fever in spite of the fact that the supposed cause of the fever—that is, osteomyelitis of the mastoid process—has been adequately drained and the local symptoms have disappeared. In this instance the examination of the urine was not made until after I saw the baby, and here, as in the first case, the urine was found to be loaded with pus.

These cases are so common that it is surprising how infrequently the diagnosis is made and how often one finds pus in the urine in the most obscure conditions. To me the most

outstanding fact in regard to these cases is that they rarely show any symptoms which point definitely to the involvement of the kidneys and bladder. In our first case there was very evidently pain in the region of the kidneys which resulted in the flexed position of the lower extremities, and pain elicited by their movements. Let us consider in a general way the question of pyelocystitis in infancy and take up some of the salient points as to etiology, diagnosis, and treatment.

Of the many causes of irregular fever in infancy few are more common than pyelocystitis. Certainly in my experience pyelocystitis is as often encountered as any cause other than gastrointestinal involvement or pneumonia, and I am not at all sure but that it is more frequent. It occurs in over 80 per cent. of the cases in girl babies, and according to statistics is most frequently met with between the ages of seven and twelve months. That there is any definite connection between diarrhea and pyelocystitis I very much doubt. It has been so frequently my experience that following operations of almost any kind temperature with pus in the urine has been encountered that I feel that the lowering of the resistance of the individual by operative procedure must have some effect on a latent infection of the urinary tract. We have repeatedly made cultures from a catheterized specimen of urine in these cases, and have in every instance but one found the colon bacillus. In this one instance, which was in a newborn baby, the culture revealed a pure growth of the *Bacillus pyocyaneus*. This may be regarded as an altogether exceptional case and we may say that in the vast majority of cases the *Bacillus coli* may be considered the cause of the infection. This has been so regularly true that unless something unusual occurs we have ceased to make cultures of the urine. How the *Bacillus coli* gets to the pelvis of the kidney is as yet undecided. Probably the majority of writers regard the ascending route as the one most likely. There is, however, a lymphatic connection between the cecum and the pelvis of the right kidney which suggests that in some instances, at least, the infection may follow this route. In certain severe cases where the kidney substance is involved it seems likely that the infec-

tion is hematogenic. I have never seen but a few of these cases go to autopsy, and the findings in these have suggested rather severe intoxication than generalized infection. The involvement of the pelvis of the kidney had been comparatively slight, but in one case which I remember distinctly the liver was so fatty as to resemble a large mass of butter.

Clinically, pyelocystitis is a protean disease and it is only to be diagnosed by examination of the urine. In the large majority of cases there is no symptom which points to involvement of the urinary tract, and it is only by routine examinations that we are able to discover these cases. At least three types are encountered, and every man who has to deal with infants should always have in mind these clinical pictures. In the first, or fulminant type, the course is very stormy. The child is taken suddenly ill with a very high temperature; the prostration is very marked and convulsive seizures are frequent. Careful examination reveals nothing. In the first twenty-four hours even the urine contains no pus, but a careful examination will reveal the presence of thousands of slightly motile, short, thick bacilli. Usually at the end of forty-eight hours pus in large quantities is found. These cases run a rapidly fatal course, death occurring within two or three days. Symptoms other than those mentioned are either absent or not suggestive. Usually in these cases we find the abdomen markedly distended, the thighs flexed, pressure in the lumbar region apparently elicits pain. Under favorable conditions deep palpation will show the kidneys enlarged. Fortunately, this is the least frequent form of pyelocystitis, but one should never forget that such a form does exist, and should endeavor to eliminate it in obscure cases of this sort.

The second type is much more usual. In this we find a baby taken suddenly ill with high fever, vomiting, and a slight diarrhea. Considering the height of the temperature, which frequently goes to 105° or 106° F., the general prostration is remarkably slight. Examination of this child reveals very little, but it is peculiar that a reddening of the pharynx is very often present. There may be some tenderness in the lumbar

region. The legs frequently in this case, too, are held flexed. The most remarkable thing about these cases is the temperature. In many instances this has a very distinctive type. A sudden rise in temperature up to 105° or 106° F. is followed by as sudden a drop, almost to normal or quite there. The result is a temperature curve with high peaks and deep valleys. There is no symptom which points to involvement of the urinary tract, and frequently on examination of the urine we are rather surprised to find a large amount of pus present.

The third type is of the sort which we encounter most frequently, and it may be said to be in the severer phases very much the same as that shown in the two cases presented. Without the temperature being so severe as to be alarming at the same time its continuous nature is such as to somewhat puzzle us, and accidentally, on a careful examination of the patient, we find pus in the urine. These children may come to us because of the temperature or they may come simply because of loss of appetite or general malaise, or perhaps even a change of disposition. Sometimes it is because the child does not sleep well.

The course of the temperature in these cases varies greatly. After the acute stage has passed children usually run a low, irregular temperature (if untreated) for days and oftentimes weeks. This may gradually disappear and the temperature remain almost within normal limits. Suddenly, without any provocation or cause which we may be able to detect, the temperature rises to 103° or 104° F., to go down again within a few hours and continue on its way. If we examine the urine during the height of the temperature or immediately thereafter, we will find an increase in the quantity of pus in the urine. This condition not infrequently accounts for nervous, poorly nourished children, and is more often seen after infancy.

The white blood count is nearly always high in pyelocystitis, and it is quite noticeable that in both our cases there is a leukopenia. This has been so frequent in all infections recently that I have been inclined to attribute it to the effect of the recent "flu" epidemic.

A few words about the urinary findings in these cases will be

of value. Practically the only finding of any importance is the presence of pus. At times it is true we do get a slight reaction for albumin; this occurs almost only in those cases where pus is present in very large amounts, or in the more chronic cases where, as happens occasionally, an involvement of the kidney tissue has taken place, and there is a reaction for albumin as the result of impairment of the kidney function. Occasionally in the acute stages quite a good many casts are to be found. In the ordinary case the examination for pus is best done without the centrifuge. A small amount of the urine is put under the low power and any large number of pus cells thus seen mean that there is an excess of these cells in the urine. In the normal urine of the infant the cell content is so slight as to be negligible, so at this stage the contrast is so marked that the condition is readily recognized. As previously mentioned, in the first twenty-four hours of an acute case the urine may show only colon bacilli and no pus cells. Occasionally, too, in the more chronic stages we find a few red blood-corpuscles. The number of pus cells in the urine as determined by the use of the white blood counter per cubic millimeter varies greatly in different cases. In the acute stages of the second type the number of pus cells is so great that it is almost impossible to count them by this method. The method is of value, however, in following an individual case, because by this means you have a fair index on the condition in the pelvis and bladder. I have usually found that where the number of pus cells is below 15 per cubic millimeter the case can readily be kept under control.

The outlook of these cases is not as good as the comparative mildness of the infection would seem to indicate. Leaving out of consideration cases of the first type, the cases of the second and third type show a marked tendency to recurrence. As a rule, the successive attacks are milder than the first, but I have seen at least two cases where the temperature went to 107° F., or over, in a second attack. One never knows when he has gotten final control of the disease. Recurrences are the rule, and happen no matter how often, or how carefully, the case is looked after. One congratulates himself that he has accom-

plished a cure, only to be met with a return of the condition in a few weeks or months.

Treatment.—The treatment in general consists in combating the *Bacillus coli*. This may be done either by the use of urinary antiseptics or by rendering the urine alkaline in order to produce a medium which is antagonistic to its growth. I have usually found that the latter method is best in the acute stages. For this purpose the child is given enough sodium bicarbonate to render the urine alkaline and keep it alkaline. To do this it is usually necessary that the soda be given in large amounts, at least 20 grains every four hours, even to young babies. It is very necessary that this be continued at regular intervals throughout the day and night; otherwise the urine becomes acid and tends to revive the infection. I used to use equal amounts of sodium and potassium salts, but I have found that in young babies the potassium is often irritating to the stomach, and have abandoned it. In babies, in order that one may know whether the urine is alkaline, it is wise to put a piece of red litmus paper between the folds of the diaper. One should always remember that ammonia formation is quite marked, and that unless the diapers are changed soon after the urination the presence of a blue piece of litmus has not the same significance that it would have otherwise. The child is kept on the alkaline treatment until the temperature has gone down and the quantity of pus in the urine is reduced. We may then switch abruptly to urinary antiseptics. I usually use hexamethylenamin, 5 grains three times a day. At the start, due to the fact that the urine becomes acid before the antiseptic action of the hexamethylenamin is present, the temperature may rise a little and the quantity of pus increase. This change for the worse, however, rarely lasts more than twenty-four hours. The hexamethylenamin is not nearly so effective as are the alkalis, and it is oftentimes necessary to switch back again to the alkalis within a very short time. By judiciously switching from one treatment to the other we usually can overcome the infection.

Other treatment of this condition is largely symptomatic. Hydrotherapy for the fever and dietetic treatment for the general

condition of the child. It should always be remembered that in young babies, and even in older children, pyelocystitis may be the cause of rather severe malnutrition, in which case dietetic measures alone are of little value.

One word in caution as to the alkaline treatment: In cases of spasmophilia the use of large quantities of sodium and potassium is contraindicated because of the tendency of these two chemicals to increase the electric irritability. I might cite here a case in point: Chrisanthia B., eighteen and a half months old, was admitted to the hospital on February 8th, and the case was soon diagnosed pyelocystitis, and the child put on 10 grains each of sodium bicarbonate and potassium citrate every four hours. On the 12th it was noticed that the child had a typical laryngismus stridulus with KOC of 3 ma. The alkalis were immediately stopped and the child put upon hexamethylenamin, with the result that the second day after the laryngismus had disappeared and the KOC was 6.5 ma.

CLINIC OF DR. SOLOMON STROUSE

MICHAEL REESE HOSPITAL

PULMONARY TUBERCULOSIS IN ASSOCIATION WITH OTHER DISEASES IN THE GENERAL HOSPITAL

Importance of the Tuberculosis Problem in the Wards of the General Hospital: Pulmonary Tuberculosis with Gastro-intestinal Symptoms. Pulmonary Tuberculosis and Hyperthyroidism. Diabetes and Tuberculosis.

THE cases which we are going to present to you today are intended to illustrate a phase of pulmonary tuberculosis somewhat different from that usually presented at a tuberculosis clinic. In the wards of the general hospital the tuberculosis problem is entirely different from the same problem in the tuberculosis dispensary or sanatorium. In the two specialistic institutions the patient is occasionally at least considered guilty before the trial and he has to prove his innocence of the disease; in the general hospital, which does not admit tuberculous patients as such, it is only too probable that in the interest attached to the disease for which the patient enters, a definite tuberculous lesion may be overlooked or a tuberculous history not obtained. Any man who sees a great number of tuberculous patients is struck by the definite tendency of such patients to hide the classical tuberculous history under the cloak of pain or other symptoms more disturbing to him than the cough. Even in frank tuberculosis the patient will deny a history of cough while at the same time he is violently coughing. If this is true with the ordinary tuberculous patients, it is markedly emphasized in the general hospital. There are two especial reasons for this difficulty, first, the patient's knowledge that a tuberculous lesion will often pre-

vent his admission to the hospital, and, second, the patient's ignorance of the importance of such a history. If he has pain in his abdomen from an inguinal hernia, or if his friends inform him that he is growing pale on account of a progressive anemia, he will tell you all about it and forget his cough; therefore, the history taker may very well overlook a very clear-cut and definite history.

From the standpoint of physical examination it is not difficult to discover the presence of tuberculous disease, but in a general hospital it is at times exceedingly difficult to differentiate between an active pulmonary infection producing symptoms and a latent tuberculous disease of no present importance. Of course, the facilities of a hospital make the ultimate diagnosis comparatively easy. Examination of chest after chest with definite pulmonary changes, evident even to a casual observer—and this condition of affairs is common enough in the general hospital—makes one at times fail to see the relationship of these findings to the patient's disease. And if we make Roentgen plates of large numbers of these patients we find the percentage of pulmonary infections unusually large. In view of autopsy statistics there is, of course, nothing unexpected in these findings, but their high incidence surely adds interest to the question of differential diagnosis and treatment.

You will note as I show the patients this morning that the point of view will be the point of view of an internist. Pulmonary tuberculosis as a diagnostic entity belongs to the field of internal medicine, and it is only the extensive sociologic and economic ramifications which put the disease in a class by itself.

CASE I.—PULMONARY TUBERCULOSIS WITH GASTRO- INTESTINAL SYMPTOMS

This young woman is twenty years of age, single, and a stenographer. She complains of an intermittent diarrhea of several years' duration. Two years ago in Canada she had some sort of febrile disease which was diagnosed as "walking typhoid," following which the diarrhea started. There has been no blood and no mucus, as far as the patient knows. She has lost steadily,

though slightly, in weight and energy. She has not complained of cough, does not expectorate, has no night-sweats, and, as far as she knows, no fever. The past history is unimportant and the family history is negative. Physical examination, as you see, shows a frail, slightly anemic young woman, with no evidence of any great loss of weight. The teeth and tonsils are negative. Just to point out positive findings, you will note as I percuss the lungs that there is slight dulness in both upper lobes, which is more marked on the right side than on the left. This dulness might be overlooked if you percussed from right apex to left apex, but it is very easily obtained if you percuss from the base up on both sides. The breathing at both apices is rough, and after cough you can hear constantly fine dry expiratory râles in the right upper. The abdominal examination is completely negative.

Laboratory Tests.—The blood examination shows a count of 4,620,000 red cells and 9200 white cells, hemoglobin 75; and a differential white count gave polymorphonuclear cells 58 per cent., small lymphocytes 24 per cent., large lymphocytes 14 per cent., transitionals 4 per cent., and no eosinophils.

Repeated examination of the stools reveal no blood, mucus, or parasites. The urine is negative. The fractional test-meal shows a steady and persistent absence of free hydrochloric acid, with the total acid ranging from 10 to 15 throughout the digestive cycle.

So far, then, the only things of importance are the pulmonary findings and the achylia. Now let us look at the temperature chart and see whether any information can be obtained from that. You will note a daily temperature variation from normal or even subnormal in the morning to 99° or 99.5° F. every afternoon. Let me call your attention to the fact that the temperature is taken by mouth. I shall not discuss diagnostic possibilities in detail in this case, because the evidence seems to point conclusively to the following course of events: The diarrhea was caused by the achylia gastrica and the achylia gastrica was produced by a low-grade pulmonary tuberculosis.

This *type* of case is rather common in the general wards of a

hospital. Masked under a symptom-complex definitely referable to the gastro-intestinal canal lies a low-grade pulmonary tuberculosis. This seems to be more common with women than with men. To this group belong those patients whose appendices are removed for indefinite abdominal disturbances, who undergo cure for gastric or duodenal ulcer, or even for nervous breakdown. In this group of patients a mistaken diagnosis is not at all difficult to make. In their history they emphasize the importance of their gastro-intestinal symptoms and forget all about a cough. For instance, our patient denied coughing, but observation showed that she coughed rather repeatedly. The gastro-intestinal history which these patients give is, as a rule, not definite and not typical of the usual gastro-intestinal diseases, as ulcer or chronic appendicitis. They may complain of gastric distress that bears no relationship to eating; or their gastric symptoms may simulate those of ulcer; they may have constipation or diarrhea; not infrequently constipation associated with much flatulence, and at times with definite pain in the right iliac fossa.

The reasons for this indefiniteness of the symptom-complex, of course, are well known. The disturbances of gastric secretion in pulmonary tuberculosis are the same disturbances that one groups under the heading of gastro-intestinal neurosis; and it is well known that the functional upset of gastric secretion may be anything from a marked hypersecretion and hyperacidity to a complete achylia gastrica. Of course, these functional disturbances are the same as may be produced by multiple other causes.

From a diagnostic point of view this type of case requires the most careful and comprehensive study of each individual in order to ascertain the ultimate cause of the symptoms. Frequently careful gastro-intestinal x-ray studies are necessary to rule out organic disturbances in the gastro-intestinal canal. In a case such as ours, where the afternoon rise of temperature indicates the presence of an infection, special pains must be taken to eliminate foci anywhere in the body and to render certain the cause and relationship between pulmonary changes and the symptoms. As a rule, when the pulmonary condition is the

cause of the symptoms, special attention to the bringing out of râles after cough will frequently clinch the diagnosis.

A word about the diagnosis of gastro-intestinal neurosis. We no longer accept such a diagnosis unless an etiologic factor is found. Formerly we were willing to rest content when we found the story of poor living, poor food, and overwork. Now, however, we believe that even these conditions are not final, and we know from experience that in many cases such conditions are simply indicators of the presence of a tuberculous lesion.

You will be interested in knowing how we are going to treat this young lady. I shall give her a mixture containing 10 drops of dilute hydrochloric acid in 1 dram of essence of pepsin one-half hour before meals, allowing her to eat freely of everything, and give her the bed-rest treatment one would give an ordinary early case of pulmonary tuberculosis.

Note (June 1st).—This patient just discussed followed instructions with a splendid result. The diarrhea has entirely disappeared. She is gaining weight. The afternoon temperature has become normal and she both feels and looks like a well woman. However, she intends to spend the spring and summer months on a farm out west.

CASE II.—PULMONARY TUBERCULOSIS AND HYPERTHYROIDISM

Now allow me briefly to refer to the second patient, who is complaining of nervousness, loss of weight, and who admits, on direct questioning, that she has been coughing. The symptoms have been present about six months. Up to that time she has had no illness which she recalls. You will note on examination a slightly emaciated young woman, the eyes are prominent, and there is a definite von Graefe sign present. The teeth are in good condition and the pharynx is normal. The thyroid is moderately enlarged. The temperature is 99.5° F., pulse 120, and there is a fine tremor to the fingers. The lungs show an area of dulness with prolonged expiration and fine dry râles over the right apex. The heart is not enlarged and there are no murmurs, but you will note from your seats the rather thudding cardiac impulse. Abdominal examination is negative and the

reflexes are all exaggerated. Various laboratory tests have been made, but furnish no information of importance for our discussion. The diagnosis is clearly incipient pulmonary tuberculosis with hyperthyroidism.

The association of pulmonary tuberculosis and hyperactivity of the thyroid gland has furnished material for medical investigation for a long time. When you stop to consider how analogous the symptoms of the two diseases may be, it does not seem at all surprising that this subject has furnished much opportunity for study. In fact, there have been men, especially of the earlier French school, who have persistently maintained that the presence of a fever in hyperthyroidism means tuberculosis. In the Great Lakes region it is not by any means always easy to differentiate the two diseases. Most of the women in this district have some enlargement of the thyroid, and except for the thyroid enlargement the other symptoms are very similar in the two diseases. The tuberculous patient is very apt to have a tachycardia, to be nervous, to have a tremor, and the eye signs may or may not be present. Consequently, we should not be surprised to find, as I have found in investigating a large number of tuberculous patients, a large percentage with all the classical symptoms of hyperthyroidism.

It seems to me that the important fact brought out by this patient is the fact of the rather common tuberculous background for cases of hyperthyroidism. Investigators are directing attention to hyperthyroidism as a symptom of focal infection elsewhere in the body, and perhaps not the least important of such focal infections will be a tuberculous lesion of the lung.

CASE III.—DIABETES AND TUBERCULOSIS

The problem of diabetes in association with tuberculosis is certainly not an easy one to solve. Even to talk about it in terms of accuracy and to attempt to draw scientific conclusions is almost impossible. Diabetes by itself is a disease the keynote of treatment of which is individualization. No two patients are treated alike, and the same patient is not treated the same way for two weeks in succession. There are all degrees of severity

of the disease, from mild to severe, just exactly as there are grades and degrees of pulmonary tuberculosis. Given three broad groupings of tuberculosis, three broad groupings of diabetes, no one of which can be sharply defined because one grades into the other, and you can easily see the possible variations of the two diseases when combined in the same patient.

From a practical point of view one need not consider those very rare cases of tuberculosis complicated by diabetes. The usual occurrence is the development of pulmonary tuberculosis on top of a diabetes. This complication is probably a good deal more common than is usually supposed, and even such an authority as Joslin has confessed to not finding either patent or latent pulmonary lesions, demonstrated at autopsy, in some of his diabetics.

We have already spoken of the large number of patients in a general hospital who show pulmonary signs which for practical purposes may be neglected. I want to emphasize very definitely that when we are treating diabetics the slightest suspicion of a pulmonary change, either active or latent, must be given the greatest respect. The modern treatment of diabetes follows the general trend of modern medicine toward prevention, and it is much easier to prevent the flaring up of a latent pulmonary lesion in a diabetic than it is to treat the same patient after the flare-up. Having personally been fooled once or twice into disregarding rather vague signs in the chest in some diabetic patients, and having seen what such disregard leads to, I at present feel more and more strongly the unwisdom and the wrong of a drastic rule of treatment of all diabetics. Perhaps one of the greatest dangers which has arisen from the wide publicity of the starvation treatment comes from ignorance of the fact that such treatment cannot be carried out in the presence of the slightest sign of lung changes without realization of the possible grave results from starvation of a latent tuberculous patient. This certainly has not been emphasized, and yet one needs to see only one or two such cases go to pieces before one realizes the truth that lies therein. I should like to briefly tell about one such case. A woman with diabetes of only moderate

intensity, of good nutrition, and of only poor finances was to see me a few years ago just at the height of the starvation idea. She wanted to get through with the treatment as soon as possible. Examination revealed an area of dulness with suppressed breathing in the right lung, but she denied any history of tuberculosis. Drastic treatment quickly reduced her sugar, but in a surprisingly short time cough developed, temperature went up, and within two weeks we were treating a case of phthisis florida. Then, too late, I found out that the patient's husband had active pulmonary tuberculosis. She died of the tuberculosis and not of the diabetes.

Given, on the other hand, a patient with diabetes and a definite but not very advanced lesion in the lung, we have certain principles of treatment which at least may guide us along the correct paths. It seems to me that the statement can be made unqualifiedly that no such patient should ever receive diabetic treatment which will permanently impair his nutrition. Despite the modern dictum that the weight chart in diabetes can be disregarded, in such cases it is particularly necessary not to disregard the weight chart. It is perfectly true that in order to maintain nutrition we must render the patient free from sugar, or at least as close to that condition as is possible. If the patient is not oxidizing his carbohydrates it is difficult to maintain nutrition, but, on the other hand, it is in many instances difficult to render him sugar free without undermining at least temporarily his nutrition. To say the least, the problem is interesting. Personally, I think the best method is as follows: The patient should be kept under observation and at rest for a period of one or two weeks, during which time relative values are established. If the temperature chart and the condition in the lungs indicate a progressive pulmonary lesion, and if the chemical work indicates a mild diabetes, our main treatment at first must be directed toward the more serious illness, the tuberculosis. An attempt at rendering the patient free from sugar is best put off until the lung condition becomes quiescent, and then only the most gradual possible reduction of diet should be practised. If such a patient can be made sugar free with a comparatively mild

reduction in his food intake he has that much better chance of subsequently oxidizing his carbohydrates, thereby improving his nutrition and combating his tuberculosis. But if, on the other hand, drastic starvation methods are necessary to render him free from sugar, then it seems to me that discretion is the better part of valor and the best we can hope for at the time is a compromise. In diabetes there is a condition of comparative well being, which it seems to me, from the clinical point of view, is often decidedly better for the patient than the theoretically scientific ideal which we sometimes aim to attain. I mean by that that some diabetic patients are better off passing a certain percentage of sugar than they are when rendered completely sugar free. This statement, I know, is considered heresy by the more modern investigators, and yet personal experience backed by conversations with excellent practitioners with years of clinical experience reveals the fact that there are unquestionably a comparatively large number of diabetics who maintain an excellent state of being and yet who continually pass sugar. I would prefer to treat my diabetic-tuberculous patient conservatively and have him run a danger, if there is one, from his diabetes, than to treat him radically and have him die of tuberculosis.

Given, on the other hand, the combination of advanced diabetes and advanced tuberculosis, and we have as hopeless a problem as can possibly confront the therapist. Scientific discussion of such a condition is impossible. The best we can hope for is comparative comfort, and the best way we can treat such a patient is to do those things which in the individual case seem most important. Symptoms are treated as they arrive, a diet is aimed at without any attempt at the metabolic treatment of a diabetic.

A CASE OF BELLADONNA POISONING

THE young lady who is now before you has kindly come from her home in order to give us the opportunity of telling you about a condition that practically is never seen in this hospital. She is married, age twenty-six, and has one child. She has always been a bright, very alert young woman, interested, as many modern young women are, in things medical and psychologic. She had always been well and active.

A few weeks ago she was suffering from a cold and the prescription which I wrote contained $\frac{1}{180}$ grain of atropin sulphate to each dose, and this was to be taken every four hours. The prescription arrived around 8 o'clock in the evening and she was immediately given one of the capsules. During the night she had been somewhat delirious, but the nurse the next morning at 7 o'clock gave her a second capsule. When I saw the patient at about 9.30 she presented a typical physical appearance of belladonna poisoning. Her pupils were widely dilated, but not completely fixed, the skin was flushed, the mouth dry, pulse rapid, and she was in the state of mental excitement and delirium which will be discussed in a moment. Her husband had also taken one of the pills, and he, too, was complaining of dryness in the throat and inability to see things at close hand. On inquiry we found that a careless drug clerk had indulged in the criminal pastime of changing a prescription, so that instead of getting $\frac{1}{180}$ grain of atropin at a dose she got $\frac{1}{8}$ grain, and in the course of twelve hours she had gotten $\frac{1}{4}$ grain of atropin.

We watched the patient very carefully for the next two hours and noted that the symptoms were gradually abating. The pulse got slower, the delirium quieted down, and the pupils became somewhat smaller. The speech and actions of our patient were interesting and apparently logical, even if disjointed. Along toward evening she was practically herself. She had realized her mental disturbance and was begging me not to send her to an insane asylum for her dementia præcox, but to give her a chance at home. The next morning she had completely

recovered, but the impressions of her mental wanderings were still so strong that I asked her to dictate everything she remembered. Now, instead of asking the young lady to repeat her confession, I am going to read you exactly as given and transcribed from stenographic notes her version made approximately twenty-four hours after recovery:

"I awakened during the night, I haven't any idea of the time. A radio-light clock was standing on the table at the head of my bed, which I thought was the light. I tried to reach it, but couldn't make my hands reach that far, so I called the nurse, who was sleeping in the same room with me, to come quickly or the light would go out. She got up and turned on the light. I heard water running as if it were pouring out of the faucets; kept seeing lightning, and insisted there was a terrible storm; saw banks of fire, but it would melt away and then I would see what the object really was. I was crazy with thirst, my tongue stuck to the roof of my mouth, my uvula seemed to be going back up my nose. I called for water, but when the nurse brought it I couldn't make my hands hold the glass. I couldn't hold anything, everything seemed to melt and slip through my fingers, and at times things were whirling around me. I seemed to be slipping out of myself sideways. There was water pouring down the walls and it was standing in pools on the floor, there seemed to be a steady stream coming down.

"The ceiling was covered with spider-webs, blowing back and forth. There was a big spider in the center of the ceiling and I made the nurse kill it before I would try to go to sleep. My hands were full of bees, some of them crawling out of their cells, and they were wiggling and squirming in my hands. I also had a wasp's nest full of wasps. (We had had an empty wasp's nest while in the country two years ago, but thought I had it with me now, and it was filled with wasps.) There were nails all over the bed and I was constantly looking for them, trying to pick them up, then couldn't find them. My hands seemed full of all sorts of things which would drop on the bed where I couldn't find them, and I kept trying to pick up the shoehorn and nail file. I thought I tore the bed to pieces looking for them. My bed was full of worms, little soft ones, crawling around, leaving lines behind them, so I kept crawling to the corner of my bed to get away from them.

"There were always figures standing around me, men and women at my bedside, and I talked with them, but what seemed

to particularly worry me was my little boy. I was afraid he would catch my cold, and I shouted for them to take him away. I saw all kinds of peculiar figures on the curtains. At times I was out of doors and saw people dressed up in my curtains walking down the street.

"I was covered with a rose-colored quilt stitched in different designs. Every little round place in the design seemed to hold a scarlet baby's face, but as I looked it seemed to melt away. I tried to pick them up and they filtered through my fingers. It seemed to be my own baby. Then I thought my baby was under the cover and was being smothered. I tried to get it out, saw it slip down between the mattress and the springs. I called to the nurse to catch it before it fell, but she did not act quickly enough, so I slipped out and under the bed after the baby. They had covered me with a white blanket with a pink border, as I had insisted that the rose-colored blanket be removed. The blanket seemed to be around the baby's neck smothering her, so I kept pulling at it; afterward the blanket seemed to be the baby herself, and the pink border was a rash on her back, and I insisted that they have a doctor see her. Later I saw her on the next bed, then the head seemed to slip off and move over to the table at my bedside, then to my own bed.

"I could just see the tips of my fingers, the rest of my hands was not visible and did not seem to belong to me. There seemed to be no connection between my finger-tips and myself. I kept trying to comb my hair and put it up, but every time I picked up a hairpin it slipped through my finger-tips.

"The nails on my bed changed to needles. I thought I had one in my foot, pulled it out, and found it was a horsehair instead of a needle.

"I was aware of some of the things that were going on around me. I spoke to my mother-in-law, who was here, and told her not to come again on account of having to climb so many stairs. I saw the doctor standing holding a match before my eyes and heard him say I had atropin poisoning. I told him I had dementia præcox.

"I realized that I was doing queer things, and kept trying not to while anyone was there, because I was afraid they would send me to an asylum. I kept thinking that if I could prove I was not crazy I would be all right, but was afraid if they once got me in an asylum I would never get out.

"I was very nervous and my hands seemed to be jumping all the time. I seemed to be on the edge of the bed, no matter how I moved. I don't know whether I really turned or not,

but thought I kept moving to the center of the bed, but still found myself on the edge of the bed. The worst sensation was when I was on the edge of the bed and couldn't reach the table, which was next to the bed.

"I tried to get the nurse out of the room so I could jump out of the window and run away. I promised not to get up and tried to send her to see the baby. The nurse stepped behind the door and watched me for a few minutes and came back and sat down near my bed; but I thought that in the short interval while she was out of the room I had been out of bed and running all over the house. When I came back into the room I saw the nurse sitting by my bed, so I slipped in bed on the other side so she wouldn't see me. Then I told her that I might as well tell her that I had been out of bed, as she would find it out anyway.

"During the night a large window appeared in the wall and I saw the shadow of a man standing there. I stared at him, thinking he would go away, then I made funny sounds, trying to frighten him. I called the nurse, she turned on the light, and then I realized that neither the window nor the man were there.

"Once I was up, dressed, and out in the street. I went over to a meeting and made a speech. I kept saying that I shouldn't be there and that my nurse would miss me. When I saw the lightning flashing I asked them if I had a tumor on my brain.

"I was sitting in a chair in the dining-room and wanted to go back to my bed, as I was very tired, but I noticed the chandelier was different than the one in our dining-room, in fact, it was like the one in my bed room, then they told me to look around the room, and I realized that I was still in bed.

"When my trays came in I found hairs in my food, picked them up, and said, "Isn't that a hair?" The nurse pretended to take it and I went on eating my food, not in the least concerned about the hairs.

"The nurse had on a white apron and every crease seemed to be a service stripe.

"I wasn't supposed to be left alone, but while my husband was staying with me I jumped out of bed, started to walk, but couldn't. I was dizzy and seemed to stagger around.

"At one time I seemed to be in Siberia. There were big banks of snow and everything was white with snow. The soldiers were camping near, then some of our soldiers came, many nurses with them, and the soldiers got down on the snow and said the Lord's Prayer.

"About 5 o'clock Monday evening I began crying to see my mother, father, and brother, but I thought it was morning."

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

MALIGNANT ENDOCARTIS OF THE PULMONARY VALVES (WITH AUTOPSY)

March 3, 1919.

THE first patient whom I wish to show you today is a colored man forty-three years of age, a janitor by occupation, who came into the hospital on February 10th, that is, twenty-one days ago. He complained at that time of cough, dyspnea, weakness, and pain in the left chest.

Onset and Course.—He states that he was sick for five days before he came into the hospital, and that the above symptoms started in suddenly and continued to increase in intensity up to the time of admission. The cough was harsh, continual, and productive—a profuse, bloody, mucoid sputum being brought up. The dyspnea was especially marked when lying down and was quite extreme. Weakness was quite marked. The pain was of a sharp sticking character which he describes as being “under the heart.” He says that it was only present when he coughed or took a deep breath.

General Symptoms.—These were, for the most part, negative. His sickness had not been preceded by any loss of weight, and he had no epistaxis, general pain, sweats, edema, or palpitation, except that he thought he had a little palpitation after considerable exertion. His appetite had been good. He had had one or two vomiting spells, but these were precipitated by the severe paroxysmal coughing spells developed at the onset. His bowels were always irregular, and he had noticed nothing abnormal with the urine.

Past History.—*Medical.*—He had pneumonia eight years ago, but does not remember on which side. He had chills and fever while living in the South a year or two ago, and was told at that time that it was malarial fever, and took quinin for it.

Surgical.—He had a fistula in ano repaired in 1897.

Venereal.—Denies all forms of venereal infection.

Habits.—Uses beer excessively, no whisky, no tobacco, or narcotics.

Family history discloses nothing of interest.

Physical Examination.—On entrance the patient seemed well developed and well nourished. He was clear mentally, with no gross deformities, and suffering principally from cough and dyspnea.

Head.—Scalp and cranium: Negative.

Eyes: Pupils regular and equal, reacting normally. Movements normal.

Ears and nose: Negative.

Mouth: Teeth show a pyorrhea. Tongue is beefy and red. Pharynx and tonsils markedly hyperemic.

Chest.—Well formed and muscular. Inspection shows considerable lagging on the left side. Percussion shows slight dulness over two or three small areas over the left lower lobe, almost flat at the base. Auscultation shows a slight decrease in the breath sounds at the base. A well-defined friction-rub can be heard on the left side anteriorly, just below the nipple. The vocal fremitus is diminished over the flat area, as is also the tactile fremitus.

Heart.—The apex-beat is readily palpable in the fifth interspace just inside the nipple line, is plainly circumscribed, and fairly forcible. Palpitation is negative. Percussion shows approximately normal boundaries, perhaps a little widened to the left. Auscultation shows the heart tones loud, of normal rhythm, and increased rate. At the apex the first tone is slightly impure, but there is no definite murmur. At the base both tones are normal and there is no abnormal accentuation.

Abdomen shows a moderate gaseous distention. There is marked diffuse tenderness over the epigastrium, but no especial

rigidity. Palpitation discloses nothing abnormal in the region of the liver, kidneys, stomach, gall-bladder, or appendix. The spleen is not palpable and shows no increased area of dulness. Genitalia are negative. Extremities negative. Reflexes are all present, but sluggish.

Blood-pressure.—Systolic, 135; diastolic, 92.

On admission his temperature was 99.2° F.; pulse, 96; respiration, 24, but as you will see from an inspection of the charts (Figs. 20 and 21), it rapidly rose to 102° F. or thereabouts. The urinalysis showed nothing of consequence except a trace of albumin. The blood examination showed a slight anemia (3,600,000 reds and 9000 whites).

I saw the case the day after admission and went over it carefully, and quite concurred in the diagnosis which my interns had made of an "influenzal" bronchopneumonia with a small fibrinous pleurisy, and probably a small amount of fluid. Our experience is that such a large number of our colored patients have tuberculosis, which in the presence of an acute lung condition may readily be overlooked, that we pay particular attention to the examination for tubercle bacilli in all such cases. In this case, however, we found no tubercle bacilli and no influenza bacilli, but nearly a pure culture of pneumococci. The patient did not seem especially sick and there was nothing about the case to stamp it as one in any way different from the large number of similar cases then in the hospital.

On February 16th, six days after admission, we have a note that his temperature is ranging around 102° F., that the physical signs in the lung were practically unchanged, and that his general condition was excellent. On the 19th his temperature had come down to normal and remained substantially normal for two days. On the 22d we find a note that convalescence was apparently satisfactory and uncomplicated, although the physical signs had not cleared up. On the 26th a careful examination showed only a few medium-sized râles remaining, with harsh breathing. The signs of fluid had disappeared. There were a few râles over the right lung as well.

Now you will note from the chart that after the temperature

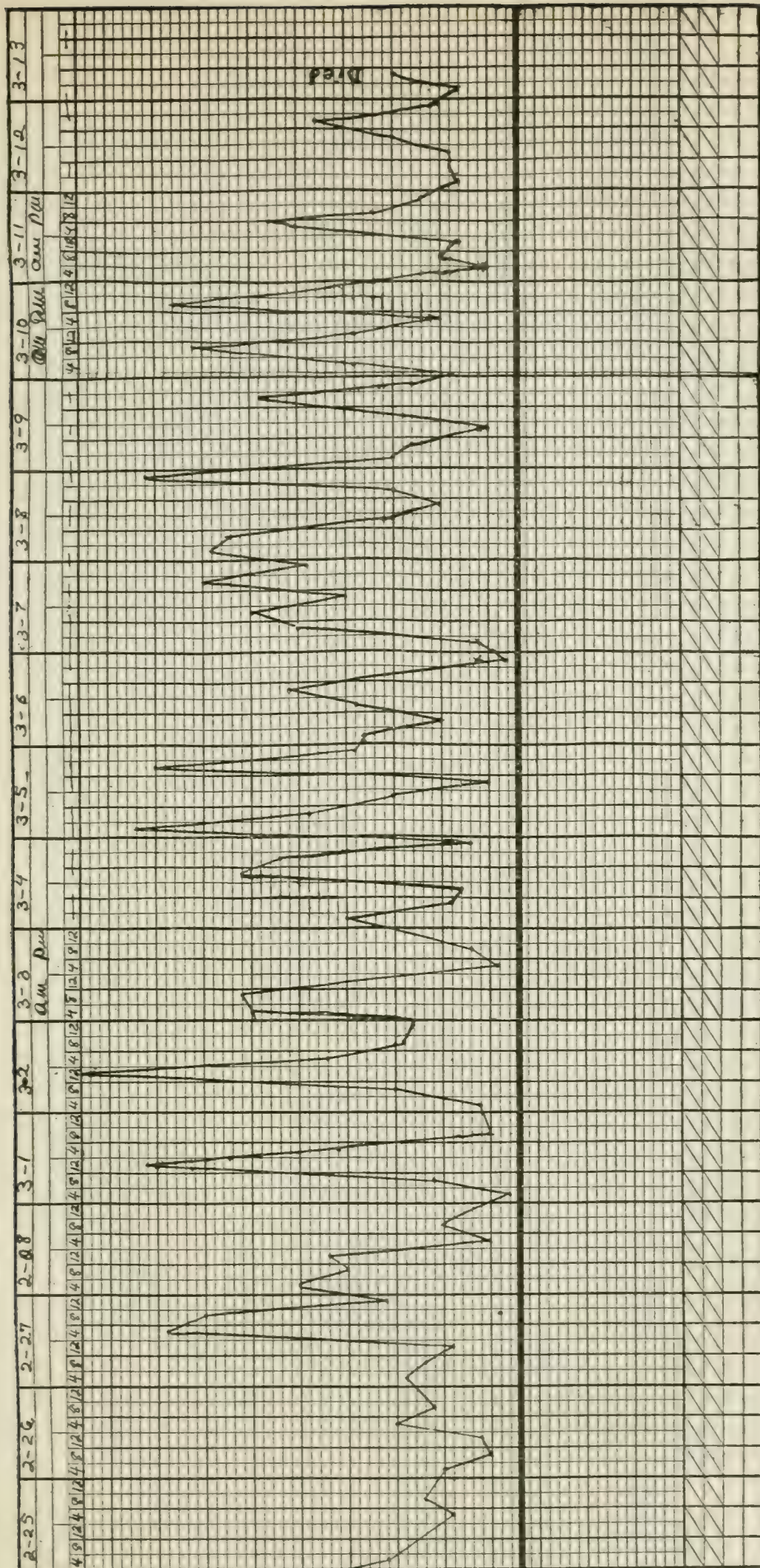


Fig. 21.—Fig. 20 continued.

had come down to normal on the 19th, it again rose to about 100° F. and remained there for a few days, reaching 102° F. on the 24th and coming down to 99° F. on the 26th. During all this time the patient seemed perfectly well and begged to be allowed to get up. The findings in the chest did not change perceptibly, and inasmuch as repeated examinations for tubercle bacilli were negative, our conclusion was that it was simply a matter of delayed resolution, of which we have seen so many. There were no areas of dulness and nothing to suggest empyema or abscess. On the 27th the patient had a violent chill, and the temperature, which at noon was 99.8° F., rapidly rose, reaching 105.8° F. at 4 o'clock, dropping again to 101° F. at midnight, going back to 103° F. the next morning, and then dropping to 99° F. that afternoon.

We went over the patient with great care, and found that the physical signs in the lungs were substantially the same, perhaps a little less marked. The heart showed a slight systolic whiff over the apex, which was not always present, but there was a very distinct reduplication of the second sound at the base, best heard over the pulmonic area. The spleen was now plainly palpable; it seemed quite hard and not tender. This was especially noted because on admission we searched particularly for an enlarged spleen, since the patient had told us that he had had malaria previously. A blood-culture was taken, which remained sterile, as did one taken the following day. The next day, March 1st, the temperature rose from 98.6° F. at 4 A. M. to 106.2° F. at noon, with a chill of great intensity, followed by a hot stage and profuse sweating. The patient, in spite of these remarkable temperature changes, felt astonishingly well and begged to be allowed to get up when his chill was over. Yesterday his temperature rose from 99.2° F. at 4 A. M. to 107.6° F. at 12, with another severe chill, followed by a drenching sweat. This morning his temperature is 104.2° F., and up to now he has had no chill. A blood count made early this morning showed a leukocytosis of a little over 16,000. The differential count showed 92 per cent. of polymorphonuclear neutrophils.

Let us go over the patient together and see what the physical

findings are now. We find the lungs showing substantially the same physical signs as they did when last noted. There are no areas of flatness or even marked dulness, no friction-rubs. The patient has just come down from the *x*-ray room, and while the written report is not yet made out, we were informed verbally that there was no evidence of abscess or empyema, and nothing suggestive of pericarditis. Turning our attention to the heart, we find the apex-beat and the boundaries unchanged. The little systolic whiff at the apex you will see still persists. At the second interspace, just at the left of the sternum, we can hear the reduplicated second sound which we heard several days ago in the same area, but much plainer. In particular I cannot make out any pericardial friction-rub. The abdomen shows absolutely nothing abnormal, except that the spleen is a little larger than when last palpated.

I think you will agree with me that the case is one of decided interest if for no other reason than for the extremely high temperature. As you will see, the patient looks in excellent condition, and still says that he has no pain and would like to get up and go home. The burning question at the present moment is as to the diagnosis. Under ordinary conditions such a temperature would suggest a severe malarial infection, and it is, of course, just possible, although highly improbable, that the pneumonic condition had stirred up an old malaria. When we reflect, however, that the patient has a very definite leukocytosis, this beomes still more improbable, and we are quite prepared therefore to find that several examinations of the blood for parasites have proved negative. Bearing in mind the great frequency of empyema, either localized or general, we have paid particular attention to the lungs, but there are absolutely no objective findings, either on physical examination or with *x*-ray, to justify such a diagnosis. Everyone who has seen many pneumonias is on the lookout for pericarditis, although this, I think, is not quite so common with these so-called "influenzal" cases as with the ordinary frank lobar pneumonia. Against this assumption we have no rounding of the cardio-hepatic angle, no increase in dulness along the left edge of the

sternum over the great vessels, no weakening of the apex-beat, and no pericardial friction-rub. On the other hand, inspection of the temperature chart almost forces upon us the conclusion that there is a septic process somewhere. The next most likely complication of this severity would be a septic endocarditis. Have we anything to indicate this? The little systolic murmur over the mitral area is still audible, but seems very trifling. When we reflect that accidental murmurs in this region are common enough in all febrile conditions, we must hesitate very decidedly to regard this as the expression of an endocarditis. On the other hand, there is another auscultatory phenomenon which has been getting more pronounced day by day, and that is the reduplication of the second tone in the pulmonary area. When we listen very intently we almost get the impression of a diastolic murmur in this locality, but this is not constant. We have searched the man's entire anatomy for evidences of localized septic process. Thinking of pyelitis, which sometimes produces a very marked septic temperature, we have looked over his urine again, but with negative results. The sputum has been examined almost daily for tubercle bacilli, bearing in mind the possibility of an extensive pulmonary involvement, like a lobar phthisis, but no bacilli have been found. The lung findings, too, make such an assumption impossible. In short, the principal feature of this case is its negative character. With a temperature range which is quite extraordinary we find almost no physical signs which enable us to localize the septic process. What, therefore, is the most likely diagnosis, all things considered? It seems to me to center in the endocardium. I am more impressed with the reduplication of the second sound in the pulmonary area than with the systolic murmur at the apex, because we have watched the former develop day by day, and it clearly represents something pathologic. It may, of course, well be that we have an old endocarditis on the mitral, with a fresh one either on the aorta or the pulmonary valve. Although the localization might very well indicate the pulmonary valve, the infrequency of this is so striking as compared with aortic lesions that we would think of the latter more especially. We

can, I think, sum up the diagnostic criteria in this case in a very few words. A patient with a clean-cut influenzal pneumonia develops a complication of the most pronounced septic type. We have ruled out malaria on the grounds already indicated. There is nothing to warrant the diagnosis of pus in the pleura, although everyone is aware how difficult this diagnosis may be. We find no evidences of pericarditis, pyelitis, or cystitis. There is nothing to point to an osteomyelitis or abscesses in the bone. There is nothing to make us suspect a brain abscess. In short, we are practically compelled to make the diagnosis of an acute septic endocarditis unless we can content ourselves with a diagnosis of septicemia. Although the signs pointing to the involvement of the endocardium are meager enough, yet there is always the one point to which I referred, namely, that the reduplication is becoming daily more marked, and in view of this fact I am going to venture the diagnosis of an acute malignant so-called ulcerative endocarditis.

We have tried very hard to cultivate some organism from the blood-stream, but as yet unavailingly. I know of nothing more which we can do to crystallize the diagnosis at the present time. I will show you the patient again at our next clinic.

March 10, 1919.

You will all recognize the patient whom we studied at the last clinic, and in whom we made a probable diagnosis of malignant endocarditis. During the past week the course has been substantially the same as when you last saw him, namely, irregular chills, fever, and sweats, with a temperature ranging from 99° to over 106° F. The most striking thing about the patient is the remarkable way in which he retains his strength. When he has no chill he wants to get up. The white blood count is steadily going up, and is now close to 25,000. The blood-culture is still negative, as is also the urine. The lungs show little change, but on examination of the heart you will hear on auscultation very definite abnormal sounds. In the second left interspace a very plain diastolic murmur can be heard, long and soft, and occasionally a short systolic blow in the same

locality. You will remember that last week we discussed at length the significance of the reduplicated second sound, and we came to the conclusion that it was of real diagnostic import because it developed day by day. This assumption, we believe, is now proved to be justified, inasmuch as the two murmurs have now developed in the same locality. This crystallizes the diagnosis of malignant endocarditis, and I think we can be quite confident of its correctness. The prognosis in this case must be regarded as highly unfavorable, although the patient's condition is apparently so good that it may run on for a considerable period of time.

Subsequent Course.—Three days later the patient went into coma rather suddenly, and died the same day.

Epicrisis.—There is nothing to add to the remarks on the diagnosis which were made at the last clinic. Just why the patient went into coma so rapidly at the last is difficult to explain. The auscultatory findings did not change in any respect, the only question which arises is as to the localization of the process. The diastolic murmur was so sharply localized to the second left interspace, as was also the systolic murmur, as to suggest a localization on the pulmonary valve. On the other hand, aortic murmurs are not infrequently heard here as well. Looked at from the standpoint of frequency, the aortic valves are involved several times as frequently as the pulmonary, and yet I think it is not sufficiently appreciated that the incidence of endocarditis on the pulmonary valve is much greater in malignant endocarditis than in the usual form. Inasmuch as the patient died before the changes in the cardiac chambers were sufficiently pronounced to be decisive, we will do well, I think, not to attempt to make the diagnosis of the exact site with certainty. Either the aortic or the pulmonary valve or possibly both would give us no surprise. One point should be especially emphasized. It not at all infrequently happens that a malignant endocarditis occurs with no murmurs at all, because of the softness of the vegetations. In this event the diagnosis from septicemia is nearly impossible unless cutaneous hemorrhages or similar embolic phenomena are present.

Summary of Autopsy Findings.—The essential thing found was a very acute endocarditis, almost entirely of the pulmonary valves. The specimen showed a vegetation of very large size



Fig. 22.—Malignant endocarditis. Large vegetation in the pulmonary valve.

in that location. A very slight older and recent endocarditis on the mitral was found. Besides a small pulmonary infarct there were no other findings of interest (Fig. 22).

GOUT

Two Cases: One Simulating a Surgical Joint Condition. Full Discussion of the Treatment of Gout, both Acute and Chronic.

THE next patient whom I want to present today is an Irish laborer, forty years of age. He was admitted with the examining room diagnosis of acute articular rheumatism. His complaints on admission related only to pain and swelling in the knees and elbows.

Onset and Course.—He states that about a week ago while he was at work he noticed some painful twinges in his right elbow. Shortly afterward the left elbow became painful, but not to such an extent as to altogether prevent his working. This condition persisted, becoming a little worse, and two days afterward both knees became stiff, somewhat swollen, and markedly painful. At no time has the pain been of extraordinary severity, but after the knees became involved he was unable to work any more, and this led him to seek hospital treatment. On admission he stated that he had no other pain except in the joints involved, and that was only of moderate severity with the joint at rest, but was very intense when the joint was moved. He volunteered the information that the pain was distinctly less in the joints which were the most swollen, namely, the knees. At no time has the swelling in the knees been marked, nor was there ever very marked redness. As he expressed it, there was “just a little red puffiness.”

General Symptoms.—His general condition had been good prior to the onset of the joint trouble. His appetite was good, bowels were in good order, though not quite regular, he had neither nausea nor vomiting, there was neither headache, cough, sore throat, excessive sweating, nor fever. He complained of no shortness of breath, but said that he occasionally had a

little palpitation when working hard. He described the urine as ordinarily normal, but had noticed that it was high colored and burned slightly since the onset of his "rheumatism."

Past History.—He had had several previous attacks of "rheumatism," the first of these a number of years ago. The most severe attack was in March, 1918, when he was in this hospital with very acute pain and swelling in the left knee, left elbow, both ankles, left wrist, the small tarsal joints, and the right great toe. (The history of that date gives a diagnosis of "acute articular rheumatism.") He had pneumonia fourteen years ago, had gonorrhea with chancroid and bubo seven years ago. The chancroid was burned off and was followed by no secondary symptoms.

Habits.—He has taken three or four drinks of whisky daily for a number of years and drinks beer also. He uses tobacco excessively, but no narcotics.

Family History.—This is negative in all respects.

Physical Examination.—This reveals a muscular, well-developed, and well-nourished male. On coming into the hospital he did not seem acutely ill, but suffering only from the pain and swelling as described. His suffering does not seem extreme, and his mental condition is clear. There are no gross deformities.

Head.—Scalp and cranium negative.

Eyes: The right eye shows an old corneal scar. The pupil is fixed and the lens shows a cataract. This, the patient states, was due to a trauma. The left eye is normal and the pupil reacts to light and accommodation.

Nose and Mouth: These are negative.

Ears: These show a number of small sebaceous cysts.

Teeth: Most of these are false, and he has a number of old stumps.

Tongue: This is somewhat coated.

Pharynx and Tonsils: These are hyperemic.

Chest.—This is well formed, expansion good, and equal on both sides. Lungs show no abnormalities.

Heart.—Apex-beat is palpable in the fifth interspace inside the nipple line. The impulse is normal in every respect. The

percussion boundaries are normal and auscultation is entirely negative.

Abdomen.—This shows a moderate adiposity and some gaseous distention, but no tumors, herniæ, tenderness, rigidity, or eruptions. Physical examination detects no abnormalities of liver, spleen, stomach, kidneys, gall-bladder, appendix, or bladder. The genitalia are negative.

Extremities.—Elbows show merely a slight reddening aside from the pain complained of, which is made much worse on motion. The knees are somewhat puffy and quite tender, but there is a fair range of motion. There is a bilateral hallux valgus, most marked on the right side.

Reflexes.—Both superficial and deep reflexes are normal.

Blood-pressure.—Systolic, 120; diastolic, 70.

The patient has been practically afebrile all the time he has been in the hospital.

Urinalysis.—The color is clear and amber, specific gravity 1010, reaction acid, and is negative for albumin, sugar, blood, and bile. On the day after admission he had 9200 leukocytes; the following day, 7200. The second day after his admission I find a note made by my senior intern as follows: "History and findings as given. Patient was in this hospital in April, 1917, with acute polyarthritis. Had a lobar pneumonia with delayed resolution while here. The recurrence now is hardly an acute attack. There is no fever and no leukocytosis. The patient should have tonsils removed and teeth fixed. A good case for vaccine treatment."

I saw this case first the day following the recording of this note, and at first glance it seemed like an ordinary chronic infectious arthritis, but there were several things that at once struck me as being out of the ordinary. To begin with, the pain, while not of great severity, seemed out of proportion to the objective findings in the joints involved. When I questioned the patient about his previous attacks of "rheumatism" he stated that he had had attacks in most of the joints, but that they had been worse in the ankles and feet. This led to a careful inspection of the ankles, and one could make out quite plainly

the residua of old inflammatory swellings there. You will notice the way in which both great toes turn out, which is noted in the history as being a hallux valgus. The patient volunteered the information that he always wore easy-fitting shoes, and that he had never had the least trouble with his great toes turning out until after he had the rheumatism there, six years ago. Inquiring then about subsequent attacks of rheumatism, he tells us that no matter in what other joint he developed the rheumatism, it always came back to his toes and ankles. The present, which is the mildest attack he has ever had, is an exception.

Now let us look at the great toes and see what we can make out of them. There is a very marked anteroposterior thickening, which at the present time is not tender except on firm pressure. It is very plain, however, that there have been very severe inflammatory processes in the joint. Examination for signs of pressure from an ill-fitting shoe, such as callosity, fails to reveal any. Now the appearance of these joints, while superficially resembling hallux valgus, in reality suggests something quite different, namely, gout. Now I do not mean to be interpreted as saying, without any further analysis of the case, that one would be justified in making the diagnosis of gout from these toes alone, and yet, I do mean, that to anyone who is on intimate terms with the clinical picture of chronic gout, such a joint as this would instantly suggest this disease.

On further inspection, we see that while the thickening of the joint from front to back is very considerable, there are no actual tophi present, in the sense that this refers to definite concretions, nor do we see any evidences of acute residual inflammatory changes there. Now, inasmuch as the presence of definite tophi constitutes the one certain pathognomonic clinical sign of gout, it is incumbent upon us to search very carefully for them. In many cases of gout of long standing the cartilages of the ears develop tophi. A reference to our history states that the ears are negative except for numerous small sebaceous cysts. Let us examine these cysts and see what they are like. We see a number of grayish-white hard nodules, several of which

are quite deep in the substance of the cartilage, others of which are more superficial. On attempting to extract one of these with a large Hagedorn needle you will observe that this is attended with some little difficulty, because they are actually in the substance of the cartilage. We have at last succeeded in getting one of these nodules out, and we will place it on the slide in a drop of glycerin and examine it under a medium power. This should, of course, be done with a minimum of light. One sees the bit of cartilage and, especially around the edges, large numbers of beautiful acicular crystals. In some places, as you will see on glancing into the microscope, there are many hundreds of them interlacing in every direction, like quills on a porcupine. Such a microscopic picture can be mistaken for nothing else. They are the typical sodium biurate crystals and are pathognomonic of gout. Now you will see from the fact that these typical tophi were mistaken for little sebaceous cysts, that it is not always easy to distinguish them macroscopically. However, close attention to the following points will generally enable you to differentiate them. The sebaceous cyst is of a more cream color, and does not seem to be so firmly embedded in the substance of the cartilage. When pricked, its contents are soft and cheesy, and not hard and chalky white. Of course, where a definite comedo can be seen, the differentiation is simple.

Under the microscope the contents of a sebaceous cyst show a mixture of oil globules with very frequently characteristic plates of cholesterol. The appearance is so characteristic that a glance generally suffices to distinguish them from each other. Yet even here it is not well to take snap judgment, for occasionally one can find more or less needle-like crystals in the matter expressed from a sebaceous cyst. These are fatty acids, but they are rarely numerous, and are never seen interlaced and felted in a piece of cartilage. I have examined within the past few days a nodule from the ear of another patient suffering from a first attack of what I believe to be gout, since he has a violent arthritis in both great toes and nowhere else, and this in a middle-aged man who has been a hard drinker is pretty surely gout,

yet on finding one tiny little nodule in his ear and removing it, oily material with a few rather atypic needle-like crystals and some definite plates of cholesterol, so what we supposed was a tophus was a little sebaceous cyst, and yet the case is, I think, one of gout. Inasmuch as it is his first attack, he has not yet had time to develop tophi. This is a good place to remind you that one quite frequently finds little fibroid nodules in the cartilage of the ear which are quite difficult to remove, and these may readily be confused on superficial examination with a gouty tophus.

To my mind the particularly instructive points in this case lie in the fact that this man has been in the hospital before with a diagnosis of arthritis, without apparently gout having been suspected. The second instructive point is that the great toes when they have been visited repeatedly by severe attacks of gouty inflammation, may be turned out in a way resembling, on superficial examination, an ordinary hallux valgus. A little attention to the shape of the joint, noting particularly the thickness in the anteroposterior diameter, should prevent this error from being made. I have here the *x*-ray plates from these toes which were not made until the diagnosis of gout had been confirmed by the examination of the tophi. The roentgenologist's report is that there are joint erosions of the great toes which he thinks might well be due to gout. I will not discuss with you here what is regarded as the pathognomonic *x*-ray picture of gout, but will merely say that they are not found in this case. In my experience the *x*-ray has not been of much service in the early diagnosis of gout, since in order to be characteristic the bony changes must have lasted for a long time indeed and have been of fairly high grade.

Just a few words about the general attitude which one should cultivate toward joint cases. With me it is a fixed rule to look at the ears carefully in every joint case, no matter what the age of the patient, and thus I have more than once saved myself from mortifying mistakes. While, as I have just told you, it is usually possible to make the diagnosis of a tophus in the ear from the macroscopic examination alone, yet, if you will follow

my advice, you will never be content with this alone. Always take out the nodule, and under the microscope the diagnosis is simple if you bear in mind the possibility of error of which I just spoke.

Our second case of gout is a private patient, a man fifty-two years of age, of whose history I shall give you merely a brief synopsis.

He comes to me primarily for vague digestive disturbances and sharp pain in the chest, having the general character of a pleurisy or an intercostal neuralgia. Incidentally he states that he has been bothered a good deal with "rheumatism," and when he came to me his right knee was bandaged, the diagnosis of chronic arthritis having been made by one of our surgeons. Beginning with his first named symptom, a careful and detailed examination of his gastro-intestinal tract failed to disclose any abnormalities, in spite of his complaint of chronic "dyspepsia." His gall-bladder and appendix are perfectly normal. He has only lived in Chicago a few months, and before that time was in New York, and being a man of large means, has invariably sought medical advice of a very high order. When he came to me an examination of the knee showed a moderate swelling which was then rapidly subsiding, the entire process being afebrile. He had had previous attacks in the knee and one severe attack in the ankle. He stated that this attack in the ankle was not really a "rheumatism" because it was brought on by a slight trauma, and his physician in New York believed that he had fractured some of the small bones of the foot, although a subsequent *x-ray* failed to reveal the postulated fracture. Further inquiry elicited the fact that he had on a previous occasion stubbed his toe slightly, following which he developed that night an exceedingly severe attack of "rheumatism" in his great toe. He subsequently, after a slight injury, developed a similar severe attack in the great toe of the other foot. With the first attack he consulted his physician, who thought it was rheumatism, and he was very carefully looked over for evidences of focal infection. When the knee

first became involved he was in Pittsburgh, and there consulted a well-known physician, who, after having x-rays made of the teeth, advised the removal of practically all of them, which was accordingly done, and following this he had no recurrence until the present attack. This was about a year ago. In the meantime, however, he has been having neuralgias of various sorts and dyspeptic disturbances, and now, since the present attack of "rheumatism" in the knee, he is utterly discouraged. His family physician has given him salicylates in large doses, and an internist called in consultation concurred in the diagnosis of "rheumatism." He was still on salicylates when he consulted me.

In regard to diagnosis, a little consideration shows that acute articular rheumatism is improbable in a man of fifty-two. A chronic arthritis of the ordinary type is far more likely, but painstaking examination failed to reveal anything abnormal in the tonsils, accessory sinuses, gall-bladder, appendix, or urethra. One might possibly have thought of the arthritis being secondary to some gastro-intestinal lesion, but we could find nothing tangible there. As his teeth had already been removed, this source of infection was ruled out. The next striking point was the fact that the toes were the first joints involved and that each attack apparently followed a slight trauma. In the only attack which I have myself observed, namely, that in the knee, the process was practically afebrile, although the joint involvement was fairly well marked. Now, after what I have already said on the subject of the diagnosis of gout, you will agree that these are points which suggest gout. When we examined our patient's ears we found a rather remarkable condition of affairs. They are thick and stiff and the larger portion of the ear is hard and infiltrated, but there are no distinct nodules apparent. On turning up the edge of the cartilage we finally succeeded in discovering a chalky white nodule the size of a pinhead, which when removed showed the most beautiful hedgehogs, made up of the typical sodium biurate crystals. They were so exquisitely typical that I have mounted them in glycerin jelly and you can see them under the microscope. You will see that there are ^Eno

fat droplets or cholesterol plates present and the crystals are typically arranged, many of them looking like needles stuck in a pin-cushion. This establishes the diagnosis of gout beyond all peradventure of a doubt. I have never seen ears exactly like these before, but I think there is no doubt that the diffuse infiltration is nothing more or less than a deposition of urates. The one little focus present escaped observation because it happened to be concealed under the overlying edge of the ear. Interpreted in the light of a case of gout his whole history is very simple. The two attacks of so-called rheumatism in his great toe were manifestly gouty. Whether or not they were actually brought on by stubbing his toe I do not know, because in many cases the patient is made aware of the fact that the joint is inflamed by a slight trauma. The painful swelling of the small joints of the ankle when he was put in plaster for a fracture, which, however, was not shown by the x-ray, when the trauma, as he describes it, was a mere trifle, was undoubtedly gout also, and both attacks in his knee, for which he had his teeth removed, were of the same nature. You will remember that tophi in the ear are the hall-marks of an old gout, and do not occur in a fresh case, so that it cannot be maintained that he has two different types of arthritis and that the gout is of recent origin. I do not suppose that anyone would seriously consider that in this case. Not less interesting are the dyspeptic disturbances and the neuralgias from which he has been suffering so acutely. When he came to me his chest had been strapped and his knee immobilized. On making the diagnosis of gout I came to the conclusion that all of his symptoms were and always had been due to this one underlying cause. I sent him to the hospital, put him on a strict purin-free diet rather low in proteins, gave him large quantities of fluid to drink, and 3 grams of atophan daily. His neuralgias disappeared like magic, and, what is more interesting, his "dyspepsia" vanished also. I have had him under observation for perhaps two months now, and, as you see, his physical condition is exceedingly good and his knee is in fine shape.

While it is difficult in this case to bring the exact proof

of the gouty nature of the neuralgias and "dyspepsia," yet the remarkable way in which they cleared up on a purin-free diet, and the fact that there has been no recurrence is to my mind sufficient justification for making this diagnosis. He is taking at the present time a diet low in purins, but absolutely no medication whatever, except an occasional day of atophan. He has just told me that he has not known what it was to feel well for ten years until he was put on a rigid diet. I am, of course, having him take as much exercise as he can, and lately he has been able to walk a good deal, which is a source of great pleasure to him. Let me again urge upon you the necessity, in middle-aged men, in looking critically at every joint case with a view to the possibility of its being gout. When we consider that this patient has twice been regarded as having a surgical condition in his joints you will see how readily this disease can be overlooked.

Treatment of Gout.—It is hardly necessary for me to premise any remarks on treatment with the statement that we shall never have a thoroughly scientific treatment of this malady until its causes are definitely ascertained. In spite of the extremely fluctuating state of our knowledge in regard to gout, there are some points which have stood the rigid test of careful clinical observation, and it is these, especially, which I want to take up with you.

The current statement in regard to gout is that it is a disease of the wealthy classes, rather than of the poor. I am not in a position to give accurate statistics along this line, but one thing is perfectly certain, that here in Chicago, at least, we see a very considerable number of cases in such hospitals as the County. I am very sure in my own mind that the idea that gout is an excessively rare disease is, for us at least, entirely erroneous. I have several cases at the present moment under observation in private practice, and there are several in the wards at the present time that I know of. About three years ago there were at one time 5 cases here on my service, and while this was very unusual, it illustrates the point that I wish to make. Now in making these statements I am leaving out of all consideration

my personal views on some of the doubtful cases, and speaking only of cases where tophi, showing the typical biurate crystals, have been demonstrated. Practically all clinicians are agreed that one of the most important—if not *the* most important—factors in the treatment is *the diet*. In choosing the diet one important thing to consider is the quantity. It might be supposed that in charity patients there would be no danger of their eating too much, but this is far from being the case, for many of these individuals are distinctly obese, and my experience has been that they are heavy consumers of beer, and not infrequently of whisky as well. It is universally admitted that alcohol is an important factor in the production of gout, but it is not usually taken into consideration how many extra calories are ingested when large quantities of beer or whisky are consumed. I do not wish to say that alcohol acts in this way only, or in this way chiefly, but this factor should ever be kept in mind in estimating the caloric intake. When possible, and with the better classes of patients it usually is possible, *alcohol in every form should be rigidly prohibited*. Of course, with the confirmed toper, this will not be feasible. *The greatest stress should be laid upon this, since, so far as my personal experience goes, it is almost a sine qua non* of successful treatment. It probably is only fair to say that this may, in a measure, be due to the fact that the patient who has not the moral courage and stamina to give up alcohol, rarely has the necessary determination to adhere to a rigid diet. The very first thing then is to select a diet which will barely suffice to keep him from losing weight, and if he is obese, as many patients are, it is rather desirable that he should lose some of the excess weight, but at a very slow rate. Sudden reduction cures are not desirable from any standpoint.

The second point in the selection of the diet relates, of course, to its composition, and in view of the theories which have been so widely held, that gout is due to a disturbance in the metabolism of the purin bodies, one would naturally jump at the conclusion that a diet rich in these would be distinctly prejudicial. Now, as a matter of fact, the exact proof that such a diet is

pernicious is, from the standpoint of physiologic chemistry, still lacking. In other words, it is not absolutely proved that when the purin intake in the food is greatly cut down the uric acid excretion will be greatly assisted. On the other hand, however, we have long known that a heavy meal, or series of meals, rich in proteins, and particularly of sweetbreads or other purin-rich meat, may precipitate an acute exacerbation of gout. I have gotten into the habit of making this clinical experiment in all doubtful cases, or rather in cases which I felt sure were gout, but in which tophi could not be demonstrated in the ears or elsewhere. On one occasion I have actually succeeded in precipitating a more or less acute attack by this maneuver, and, of course, while this was far from being an accurate demonstration, that it was the purin content of the food which induced the attack, nevertheless it is a thoroughly reasonable assumption and one quite in accord with the observations of clinicians of large experience. This seems to me the very best reason for instituting a diet very low in the purin bodies. My experience leads me to agree with those authors who use this method of treatment, and my therapeutic successes have all been in patients who could and would adhere to this line of treatment. It is, of course, a practical impossibility in a diet which is at least semipermanent, if not permanent, to exclude the purins absolutely, since even vegetables, which are relatively low in purins, nevertheless do contain some, although, of course, they are low as compared with the meats. I have had placed on the blackboard a table of the purin content of many foods, condensed from Brugsch and Schittenhelm, after Schmid and Bessau. The table on page 143 shows that the highest purin values are given by calves' sweetbreads (thymus), beef liver, beef kidneys, tongue, and squabs, and in a general way by all the different varieties of meat. A glance at the figures for the different varieties of fish and shell-fish shows that anchovies, sardines, and herring contain large quantities of purin, while many of the other fish contain fully as large amounts as beef. The shell-fish contain somewhat less, and yet oysters contain a very respectable amount. It is an assumption commonly made that a vegetable diet con-

PURIN CONTENT OF SOME FOODS

(Adapted from table of J. Schmid and G. Bessau)

Meats.

Beef.....	0.111
Veal.....	0.114
Mutton.....	0.078
Pork.....	0.123
Cooked ham.....	0.075
Raw ham.....	0.072
Tongue (calves').....	0.165
Liver sausage.....	0.114
Brunswick sausage.....	0.030
Salami sausage.....	0.069
Blood sausage.....	0
Brains (hog).....	0.084
Liver (beef).....	0.279
Kidneys (beef).....	0.240
Sweetbreads (calves').....	0.990
Lungs (calves').....	0.156
Chickens.....	0.087
Pigeons.....	0.174
Geese.....	0.099
Venison.....	0.117
Pheasant.....	0.102
Bouillon (100 gm. beef cooked for two hours)....	0.045

Fish.

Haddock.....	0.117
Cod.....	0.114
Eel (smoked).....	0.081
Salmon (fresh).....	0.072
Carp.....	0.162
Perch.....	0.135
Pike.....	0.144
Red herring.....	0.084
Herring.....	0.207
Trout.....	0.168
Sprats.....	0.246
Sardines (oil).....	0.354
Anchovies.....	0.465
Crabs.....	0.060
Oysters.....	0.087
Lobster.....	0.066

Vegetables.

Cucumber.....	0
Lettuce.....	0.009
Radishes.....	0.015
Cauliflower.....	0.024
Curled cabbage.....	0.021
Chives.....	a trace
Spinach.....	0.072
Carrots.....	0
Green cabbage.....	0.006
Brown cabbage.....	0.006
Kohlrabi.....	0.033
Celery.....	0.015
Asparagus.....	0.024
Onions.....	0
Green beans.....	0.006
Potatoes.....	0.006
Tomatoes.....	0

Legumes.

Green peas (fresh).....	0.081
Dried peas.....	0.054
Lentils.....	0.162
Beans.....	0.051

Sundry Articles.

Mushrooms.....	0.015
Cream cheese.....	0.015
Cheese.....	0
Milk.....	0
Fruit.....	0
Eggs.....	0
Caviar.....	0
Cereals.....	0
Bread (white).....	0
Nuts.....	0

N. B.—The values are all expressed as uric acid.

tains negligible amounts of purin. A reference to the table shows that this is more or less true of many vegetables, with, however, certain conspicuous exceptions. You will see that spinach contains a considerable amount, the values for this vegetable being the same as ham. The legumes are, however, the chief offenders in this respect. *Lentils*, which are so extensively used by many of our foreign-born citizens in soups, and which are, I think, coming more and more into general use as an article of diet, have a high purin content. *Peas* and *beans* also contain considerable quantities, so that when on a strict diet these vegetables should be entirely excluded.

The basis of our diet, therefore, in the beginning at least should consist largely of cereals, milk and eggs, fruit, and the vegetables which are low in purin content, excluding the legumes and spinach. The ordinary white bread or cornbread is permissible. Some authors, notably Gigon, limit the bread intake on theoretic grounds to a minimum amount, and believe that its place can be advantageously taken by potatoes. I am accustomed to allow ordinary white bread quite freely, and it is very questionable whether the theoretic grounds on which bread is forbidden are sound. Fats of all sorts may be freely allowed; this is especially true of cream and butter. It occasionally happens that a thin nervous gouty person is made distinctly worse by injudicious "dieting," and under these circumstances a liberal diet, especially rich in milk, cream, and butter, is of distinct value.

It is a debatable question as to just how long such a diet should be continued. It can certainly be kept up for many months with great advantage. Just before being called into the service during the present war I placed a patient who was suffering from a typical toe-gout, of which he had had numerous severe attacks, upon a rigid purin-free diet, or, more properly speaking, purin-low diet, to which he has consistently adhered for practically two years, and he has had no attacks since. Substantially no other treatment was followed than that the total amount of food was lessened, and he has taken considerably more exercise.

Mineral Waters.—Volumes have been written on the action of mineral waters in gout, and almost all varieties have been recommended. It seems certain that the benefits which undoubtedly accrue from treatment in some of the spas depend upon the collateral treatment, such as increased exercise out of doors, a simple and well-regulated diet, absence of factors conducive to nervous stress and worry, and last, but by no means least, to the fact that large quantities of fluids are taken. It may be that the undoubted good effects of residence at these spas upon the gastro-intestinal tract may account for some of the improvement.

Fluids should be taken with great freedom except when contraindicated by coexisting cardiac conditions. It is highly probable that it is a matter of indifference what kind of water is drunk, provided only that it be taken in sufficient quantity. I urge you strongly to give the patient definite instructions in this respect, since a mere suggestion to drink plenty of water means little to him, and is not likely to be productive of results. Coffee and tea are, of course, forbidden, except that a caffein-free coffee may be permitted.

Experience has shown me that there are several respects in which highly intelligent patients are likely to make an error. One of these is in connection with soups. I permit vegetable and cream soups, except such as contain legumes. If you do not especially warn the patient, he is likely to interpret this as meaning that all soups are allowable, whereas, soups made with meat stock or bouillon or containing extractives in considerable amount are forbidden.

Exercise.—This, of course, can only be carried out in the intervals between attacks and in the cases where the toes have not been so badly involved as to interfere with walking. In spite of the fact that the necessity for vigorous exercise in those predisposed to gout has long been recognized, I think it is not sufficiently insisted upon, although it is not perfectly clear in what way it is beneficial. The mistake should not be made of attempting to make a patient walk where the toe joints, or mayhap the ankle too, shows anatomic changes which make this un-

desirable. In a general way any outdoor sport suitable to the age, muscular development, and habits of the patient may be recommended. In our large cities this securing of sufficient exercise is a matter of considerable difficulty, and it will require a good deal of determination and enthusiasm on the part of both patient and doctor to secure the desired result. You will have especially to combat the idea, unfortunately so prevalent among our well-to-do classes, that it is perfectly satisfactory to take almost no exercise eleven months in the year and then to subject one's self to very strenuous exercise during the remaining month.

In a clinical lecture of this sort it is hardly possible to enter upon the different forms of exercise, with any discussion as to their relative merits, but I cannot pass the matter by without expressing my opinion that there are few places where the interested practitioner can do his patient a greater service than by instructing him along these lines, and by arousing enough enthusiasm in his patient to carry it out. In these days where the motor vehicle threatens to make the average business man forget what his legs were made for, it behooves us, as physicians, to insist that our patients get an adequate amount of exercise, preferably, of course, in the great out-of-doors, and that this exercise be taken systematically and regularly. The layman, not without some truth, is apt to state that the physician always recommends to his patients to take such exercise as is agreeable to the physician himself. The matter is of sufficient importance to merit a little painstaking inquiry. One man can be induced to walk to his office, another can be induced to play golf or tennis with sufficient regularity. Some may find it possible to ride horseback; a more vigorous individual will find handball agreeable; still another may take up gardening, and if all other courses fail, it is sometimes possible in our large cities to send one's patients to professional trainers for an hour's daily exercise. This latter course often falls down because the patient considers it drudgery, but, especially during the inclement months, it frequently seems the only thing available. A very few patients can be induced to take setting-up exercises with profit. If I

seem to emphasize this subject unduly, it is because the doctor himself is usually among the worst of sinners as regards lack of exercise, as anyone who has had much experience in our army training camps can abundantly testify.

Treatment of an Acute Attack.—The first thing to do is to put the patient at rest in bed. The joints should, of course, be immobilized and kept warm with cotton batting. I have used a kaolin cataplasm with gratifying results. It is an old and, I think, thoroughly sound clinical observation that cold compresses are bad. If the patient is known to have gout I instruct him to take atophan at once, $\frac{1}{2}$ gram five to eight times a day on the first day, and four times a day thereafter during the duration of the attack. I prefer, if there is any considerable fever, to keep the patient upon a milk diet, to which cereals and fruit may be added if it seems desirable. In some cases atophan works like a charm. It is certainly as good as colchicum, and has one decided advantage, namely, that its strength can be depended upon. I have never observed the severe symptoms which have been described by some authors, such as headache, diarrhea, vomiting, etc. Just how atophan acts is still a matter of discussion, but that it does increase the uric acid excretion very materially seems to have been demonstrated beyond a reasonable doubt. It has been recommended to give sodium bicarbonate simultaneously with atophan in sufficient amount to definitely alkalinize the urine. This recommendation is based upon the observation that where gout is complicated with kidney stone, an attack of colic has been precipitated by the atophan alone. I am in the habit of prescribing it with sodium bicarbonate in equal amounts. The so-called alkaline treatment, formerly so much in vogue, would best be abandoned. It is hardly necessary to say that large quantities of fluid should be given during the acute attack as well as in the interim.

It is occasionally stated that atophan has a specific action in gout. This is certainly not true so far as the mere improvement in the clinical symptoms is concerned, for I have repeatedly observed almost as marked improvement in cases of arthritis which were surely non-gouty. The diagnosis *ex juvantibus* is

certainly not permissible. The continuous administration of atophan is not to be recommended. Where there have been repeated subacute attacks the patient should be instructed to begin the régime mentioned, and the atophan at the first twinge, and never to let the attack become severe before initiating the treatment. Some authors give larger doses than those I have mentioned, but I have not found this necessary.

Colchicum.—This, so far as my experience goes, is the only other drug worthy of mention, and for generations it has been regarded as a specific. When used at all, it should be employed in adequate dosage and for a short time only, since its action is generally manifested within twenty-four hours. I have observed very great differences in the intensity of the action in both the wine and tincture of colchicum obtained from different sources, and on several occasions have seen serious toxic symptoms. Because of this uncertainty of action it is distinctly preferable to use the alkaloid, and I have used it in this form with great satisfaction, giving a dose of 1 milligram five or six times a day during the first twenty-four hours of the attack. The use of colchicum rests upon purely empirical grounds, but I think it will not be doubted by any one who has used it in a severe acute gout that its action is little short of specific.

Other Drugs.—A considerable number of drugs have been used and recommended as so-called solvents of uric acid. None of these have stood the test of experience, and their discussion is, therefore, superfluous.

Radium.—After recommendation by the German school, radium has been used to a considerable extent. I have had no personal experience with this treatment.

To sum up, while it is probably true enough that a case of gout is never actually cured, there is, in my judgment, no room for the prevailing pessimism regarding its therapy. When recognized early or, indeed, as in the case I have just shown you, even when it has existed for years, appropriate treatment may lessen the number and severity of the attacks in a way that is most satisfactory.

CLINIC OF DR. PETER BASSOE

PRESBYTERIAN HOSPITAL

THE SWIFT-ELLIS TREATMENT OF PARETIC DEMENTIA

TODAY it is my purpose to bring before you a number of patients, to relate briefly histories of other patients, and to present statistical data and charts in order to enable you to draw your own conclusions regarding the value of a much contested method of treatment for a disease of the brain, the seriousness and utterly sinister prognosis of which when allowed to take its own course are universally recognized. The disease referred to is paretic dementia, also known as general paralysis of the insane, and the treatment to be discussed is that introduced by Swift and Ellis, namely, combined intravenous injection of salvarsan and intraspinal injections of "salvarsanized serum." The history of the development of this treatment is too well known to all of you to require repetition. It will suffice to recapitulate the two chief groups of facts and theories on which the theoretic basis of the treatment rests:

1. The evidence in favor of the superiority of intraspinal or combined intravenous and intraspinal administration of remedies in diseases of the central nervous system:

(a) The recognized clinical fact that meningitis and tetanus serums are much more efficient when administered intraspinally.

(b) The recent work of Weed,¹ Frazier,² and others on the origin and circulation of the cerebrospinal fluid, particularly the demonstration that vital stains injected intraspinally reach the pial surfaces of the brain and also penetrate the cortex, while stains injected intravenously only penetrate the pial

¹ Weed, Jour. Med. Research, 1914, p. 31.

² Frazier, Jour. Amer. Med. Assoc., 1915, p. 64.

meshes sparingly and not at all the cortex. It seems that the villi of the choroid plexus and the walls of the cerebral capillaries prevent penetration of dyes and medicinal substances in sufficient quantity to be of great therapeutic value.

2. Evidence in favor of the therapeutic value of the "salvarsanized serum":

(a) The original demonstration by Swift and Ellis¹ that such serum has greater inhibitory action on cultures of the *Spirochæta pallida* than normal serum or the serum of syphilitic patients, and that this action is at its maximum one hour after intravenous injection of salvarsan.

(b) The demonstrated presence of minute quantities of salvarsan or its derivatives in the serum. The average result of a large number of analyses tabulated by Swift indicates an average of 0.016 mg. salvarsan per cubic centimeter of serum.

(c) The possible but not demonstrated presence of syphilitic antibodies in the serum.

(d) The possibility of the curative value of other unknown constituents of blood-serum as indicated by the unexplained success of serum injections in certain skin diseases, such as psoriasis, dermatitis herpetiformis, and more recently of intraspinal ones in Sydenham's chorea.

However, at least part of the success of any form of intraspinal injection method regardless of the substance used may be due to the production of an irritative process, with hyperemia and increased permeability, allowing greater penetration of the salvarsan or other useful substances present in the blood.

The patients to be presented are selected from a series of 26 cases of parietic dementia in whom the treatment was commenced in the years 1914-16. Eight of these patients are known to be alive at the present time (May, 1919), and 3 of them are mentally and physically apparently well, 2 are mentally well, but have acquired a spastic paraplegia, largely as an untoward result of the treatment. One, a case of taboparesis, is mentally perfectly well and serologically negative, but is crippled on account of an ununited painless fracture of the neck of the femur.

¹ Swift and Ellis, Archives of Int. Med., 1913.

One remains able to work, though he shows some mental impairment. The eighth patient was temporarily improved, but recently had several apoplectiform attacks, and is now utterly demented and bedridden.

Fifteen of the 25 patients are known to be dead. In one of them, however, the disease apparently was arrested, and he returned to work for a period of three years, then became ill and died, cause unknown. One patient, a physician, had a partial remission of over two years, then developed a spastic paraplegia, declined mentally, and finally died. A few of the others showed temporary improvement, while in some patients the treatment had no effect.

Our first patient is the one grouped as still able to work, though showing some mental impairment. He has received the largest number of intraspinal injections (thirty-three) of any of the 26 cases, and once he passed through a severe meningeal reaction ("aseptic meningitis"), from which he recovered without sequelæ.

CASE I.—*Syphilis in 1893. Transient aphasic attacks in December, 1912, March, 1913, and May, 1914. Nervousness and failing memory for one year before treatment commenced. Rapid improvement. He has worked most of the time since August, 1914. January, 1916, transient confusion. September, 1916, meningeal reaction followed by temporary sphincter disturbance. Thirty-three spinal injections.*

The patient, a clerk fifty-three years old, was referred by Dr. B. C. Corbus. He gives a history of chancre and secondaries in 1893. He then had mercurial inunctions for two years and had one dose of salvarsan in 1912. His wife has tabes. He has been temperate and a steady worker, having held the same position for thirty years. He was somewhat nervous for six months prior to the first aphasic attack, which occurred suddenly while he was at dinner in December, 1912. He was unable to speak for half an hour. A similar attack occurred three months later, and a milder one a week before his first admittance to the hospital on May 27, 1914. The last attack was preceded by three weeks of headache, and his wife had noticed that his memory

had been poor for a year. He had lost 25 pounds in weight during the preceding two years.

On examination, it was found that both pupils, especially the left one, reacted very little to light. The tendon reflexes were barely obtainable on reinforcement. The speech was slightly slurred and he had difficulty in repeating test sentences. The blood-pressure and urine were normal. He remained at work and after the third treatment marked improvement set in. He took a vacation in July, 1914, and since August of that year he has been steadily at work, not even losing any time on account of the treatment, as he came to the hospital on Saturday afternoon for the intravenous injection, received the intraspinal one on Sunday, and returned to work Monday morning. In November, 1914, it was noted that the pupils reacted better to light, but the knee-jerks were not obtainable. It will be seen from the table that he had no injection for nearly five months following the one in August, 1914, and by the end of this period he again became nervous, his speech was a little slurred, he sometimes could not find a word, and he complained of paresthesias in the left side. A few days after the next injection his speech again became normal and he was less nervous. He was in excellent condition during the remainder of the year except that in the latter part of December he displayed a little nervousness and occasionally complained of headache.

When examined just before his injection on January 9, 1916, both pupils reacted a little to light, the right knee-jerk was absent, the left one very weak, the ankle-jerks were absent; the wrist-jerks were weak, as was the left elbow-jerk, while the right one was not obtained. Mentally he appeared perfectly normal and his speech was entirely free from impediment.

The Wassermann test with the blood has fluctuated in a marked manner, and the same is true of the gold test, although there has been a general tendency to a lighter reaction in the latter.

He was so well for the next two months that he would not take time for any other treatment than mercurial inunctions. On the morning of March 3, 1916, he left his home as usual, com-

plaining only of headache, but did not arrive at his place of business. At 9 A. M. he was found in a dazed condition lying on the floor in the corridor of a neighboring office building and taken to a hospital. There he became excited and somewhat violent. When identified his wife and son were summoned, but he did not recognize them, was restless, and fumbling. There were no new physical findings. The next day he was calm and oriented, but did not remember the events of the previous day, and was perplexed about being in the hospital, as he remembered having an appointment for two weeks later. On account of his restlessness it was impossible to give him an intravenous injection, and he was put to considerable pain in connection with the attempt. The following day, March 5th, he did not remember that anything had been done to his arm, but was otherwise clear. On March 10th he went home, apparently as well as usual, but five days later he became restless. When readmitted to the hospital on March 24th he was slightly confused and remained so to a slight degree for a month, then appeared perfectly normal and returned to work on May 6th.

Everything progressed most favorably until a few days after the last intraspinal injection on September 10th. He left the hospital in excellent condition the next day, but a few days later headache set in and he had difficulty in voiding urine. He returned to the hospital on September 16th and had a chill, followed by temperature of 103° F., with subsequent fall to 98.4° F. He rested quite well during the first part of the night, but on the morning of the 17th he had to be catheterized, 1260 c.c. of urine being obtained. He then had to be catheterized at least once daily for five weeks, and gradually developed cystitis, which later rapidly improved on appropriate treatment, with gradual return of normal bladder function. At times he complained of severe headache and there was some stiffness of the neck. The temperature remained normal after September 22d. On September 19th lumbar puncture was made. The fluid was cloudy, contained 1992 cells per cubic millimeter, largely polymorphonuclear, but no bacteria were obtained in smears or cultures. He left the hospital on November 18th,

but soon afterward found some difficulty in controlling his rectum. By the use of daily oil enema and internal administration of bismuth subnitrate this trouble subsided in a week's time, and he resumed his work, apparently no worse for his peculiar reaction.

The next lumbar puncture was made on January 27, 1917. The cell count was then only 3 and the Nonne and Lange tests were negative, showing that the meningeal irritation had entirely subsided. The Wassermann test was positive with both blood and spinal fluid. During the summer of 1917 he was feeling well, but lost considerable weight. He was given several intravenous injections of novarsenobenzol, with spinal drainage, and spinal injections were resumed in July, 1917. It will be noted from the table that the fluid obtained on October 6th contained 27 cells and gave a typical paretic curve with the Lange test. The same was true of the fluid secured on February 2, 1918. His clinical condition, however, was good. In March, 1918, there was a temporary return of speech disturbance and difficulty in naming objects. At this time an intraspinal serum injection was given, which was followed by a brief reaction, consisting of pain, vomiting, and elevation of temperature to 100.6° F. When he returned to the hospital three weeks later he had forgotten his pain and thought he had been in the hospital only two days, while he really had stayed five days. No reaction followed the intraspinal injection on March 24th, nor the following one given on April 25th. On June 17th while at work he seemed dazed for a few minutes and came to the hospital at once. On account of difficulty with the veins, no intraspinal injection was given until September, and this time no reaction occurred except a brief rise in temperature. From this time on all of the treatments were uneventful except that half an hour after an intravenous injection on February 5, 1919, he had an attack of trembling, which lasted eighteen minutes, during which he muttered incoherently. On account of the condition it was impossible to secure blood for intraspinal injection. The next day his mental condition was as usual. At the time of the treatment in March and April he was feeling very well and had gained

in weight. However, he showed unmistakable evidence of a mild deterioration and lack of insight. It had been necessary at his place of employment to give him simpler work than he

CASE:	DATE	WASSERMAN : BLOOD	WASSERMAN : SPINAL FLUID	CELLS : GLOBULIN TEST	NONNE-APERT :	LANGE GOLD TEST	INTRAVENOUS : Neosalvarsan :	INTRASPINAL : S.E.C. :
1	1914							
	May 29	+	++	76	++	3 4 4 4 3 3 2 1 0	.45	: 30 : 40 pet.
	June 13		++	15	++	5 5 4 3 5 4 3 1 1 0	.7	: 20 : 50 pet.
	July 2		++	10	++	5 5 5 4 3 3 3 2 1 0	.9	: 25 : 40 pet.
	" 29	+	++	12	+	1 1 2 2 1 1 0 0 0 0	.9	: 20 : 30 pet.
	Aug. 13	+	++	12	+	1 2 3 3 1 0 0 0 0 0	.9	: 30 : 50 pet.
	1915							
	Jan. 14	++	++	17	+	5 5 4 4 3 0 0 0 0 0	.9	: 20 : 60 pet.
	" 21	-	++	13	+	5 5 5 4 1 0 0 0 0 0	.9	: 30 : 60 pet.
	Feb. 21	-	++	13	+	5 3 3 1 1 1 0 0 0 0	.9	: 32 :
	March 21	-	++	9	+	2 3 3 2 1 0 0 0 0 0	.9	: 28 :
	April 18	+	++	7	-	0 1 1 1 0 0 0 0 0 0	.9	: 32 :
	May 14	++	++	13	+	4 4 3 3 2 1 0 0 0 0	.9	: 30 :
	June 13		++	14	+	1 1 2 2 2 2 1 0 0 0	.9	: 35 :
	July 20	-	+	4	-	1 2 3 3 3 3 1 0 0 0	.9	: 37 :
	Sept. 12	?	-	4	-	1 2 2 1 0 0 0 0 0 0	.9	: 33 :
	1916							
	Jan. 9	+	++	24	++	1 3 3 4 3 2 2 0 0 0	.6	: 42 :
	March 9	-	++	4	+	1 3 4 4 4 3 1 0 0 0	As.B.4	: 30 :
	" 27						As.B.4	:
	April 3						As.B.6	:
	" 20	-	++	4	+	1 3 4 4 4 3 1 0 0 0	As.B.6	: 42 :
	May 3	++	++	7	weak	1 2 1 0 0 0 0 0 0 0	As.B.6	: 40 :
	" 27	++	++	2	+	2 2 1 1 0 0 0 0 0 0	As.B.6	: 35 :
	June 25	++	++	1	+	2 3 2 1 1 0 0 0 0 0	.9	: 32 :
	July 30	++	++	8	++	2 1 1 1 0 0 0 0 0 0	.9	: 40 :
	Sept. 10	++	+	5	-	1 1 1 1 1 0 0 0 0 0	.9	: 32 :
	" 19		++		++	Fluid cloudy. No bacteria.		:
1	1917							
	Jan. 29	++	++	3	-	0 1 1 1 0 0 0 0 0 0	.9	:
	April 21	++	++	15	+	2 3 3 3 1 1 0 0 0 0	.75	:
	July 5	-	++	7	+	1 2 1 0 0 0 0 0 0 0	As.B.6	: 20 :
	Oct. 6	++	++	27	+	5 5 5 5 4 4 3 1 0 0	.9	:
	Oct. 27						.9	:
	1918							
	Feb. 2		++	25	+	5 5 5 4 3 2 1 1 0 0	As.B.6	:
	March 3	+	++	24	+	5 4 3 3 3 2 1 0 0 0	.9	: 20 :
	March 23						.75	: 15 :
	April 24	-	++	6	+	2 3 4 4 2 2 0 0 0 0	.9	: 25 :
	June 22		++	10	+	1 2 2 3 3 2 1 0 0 0	.5	: 25 :
	July 16						.75	:
	Aug. 17						.75	:
	Sept. 18	+	++	15	+	1 2 3 3 2 1 0 0 0 0	.9	: 25 :
	Oct. 31	++	++	13	+	2 3 3 3 2 1 1 0 0 0	.9	: 22 :
	Dec. 2		++	15	+	4 5 5 5 3 2 1 0 0 0	.9	: 25 :
	1919							
	Jan. 5						.9	: 20 :
	Feb. 5						.6	:
	March 22						.75	: 32 :
	April 23	+	++	16	+	1 2 3 2 1 1 0 0 0 0	.75	: 25 :

Table I, Case I.—As.B. means arsenobenzol. On all other occasions neosalvarsan was given. When no percentage is stated the serum was not diluted with salt solution. ++ means strongly positive reaction.

had been accustomed to. This change of work seemed to puzzle him, but he did not realize its true meaning.

Summarizing this case, we have here before us a man who had characteristic apoplectiform attacks causing transient

aphasia as early as 1912, and in 1919 he is still able to earn his livelihood. He has less speech disturbance and is less nervous and irritable than he was at the beginning of the treatment, but a mild and probably permanent deterioration of the higher mental faculties has taken place. It is not going too far to assert that without treatment he undoubtedly would have been completely incapacitated several years ago and probably dead. The immediate beneficial effect of the intraspinal injections was apparent on a great many occasions, and some of the worst relapses occurred when no injections had been given for several months. The extremely severe reaction which followed the treatment in September, 1916, is of the type described as "aseptic meningitis" by Henry A. Cotton¹ in his exhaustive paper on the Swift-Ellis treatment.

CASE II.—Tabes for at least one and one-half years. Then became nervous, irritable, made mistakes in calculation, trembled in writing, depressed, and worried. Ordinary physical signs and spinal fluid findings of tabes. Steady improvement with Swift-Ellis treatment. Resumed work in five months and appears normal mentally.

An insurance clerk, forty-one years old, was referred by Dr. Glenn Wood on April 14, 1916. History of syphilis twelve years previously. Patient has never used alcohol, but formerly smoked considerably. One and a half years ago he noted uncertainty on his feet and had slight urinary incontinence for a time. A year later he developed lancinating pains in the legs. Numbness in the left foot and unsteadiness became so marked that he fell on several occasions. Impairment of memory and decreased mental capacity had been noted for about six months and for some weeks his writing had been tremulous. He was nervous, irritable, and slept poorly. Between August, 1914, and March, 1915, he had received six injections of neosalvarsan and also had been treated with mercurial injections up to November, 1915.

Examination on April 14, 1916.—Argyll Robertson pupils.

¹ Amer. Jour. of Insan., July, 1915.

Knee- and ankle-jerks absent. Marked ataxia of legs. Tremor of hands. Analgesic areas on chest and arms. He calculated quite well, but could not repeat test sentences.

On April 7th he received arsenobenzol 0.4 intravenously. On April 22d intramuscular injections of salicylate of mercury were commenced. When seen on April 26th he was worse mentally, in constant fear of the police, and imagined he had to have their permission to go out. Soon after the first intraspinal injection on May 1st he began to improve, and in another two months he became quite cheerful and perfectly clear. The ataxia improved and the numb feeling in the legs disappeared. He returned to work in September, and his employer informed me on December 15th that his work was very good.

The changes in the laboratory reactions are noteworthy. It will be seen from the table that all the tests were positive at first, and that those made on August 11th were entirely normal. Later the tests again became positive, although the patient continued to improve.

He was given mercurial inunctions between the salvarsan treatments, and continued to do well until December 18, 1917. He then fell on an icy sidewalk, but felt no pain, walked home, and upstairs. An hour later when he raised a window he felt something snap in his right hip. He was brought to the hospital the next day and was then able to raise the right leg and hold it up. Nevertheless, x-ray examination showed an intracapsular fracture of the femur. Dr. Phemister made a bone peg from the patient's left tibia and drove it through drill holes in the two fragments. The operation, however, was not successful, possibly partly because the cast was softened by the patient's urine, which was voided involuntarily. He developed a severe cystitis, with considerable fever for several weeks, and during this time he was confused mentally. However, after a few months the cystitis cleared up, he walked well with crutches, and appeared entirely normal mentally. He returned to work in his former position and remained at work. As will be seen from the table, the last laboratory tests made in January, 1919, were entirely negative.

We here are dealing with a case of tabes which became one of taboparesis, and now, apparently as the result of treatment, again is one of tabes only.

CASE:	DATE	WASSERMANN:	WASSERMANN:	CELLS:	NONNE-APELT:	LANGE	GOLD	TEST	INTRAVENOUS	INTRASPINAL:								
:	:	BLOOD	SPINAL	:	GLOBULIN	:	:	:	NEOSALVARSAN	SERUM								
:	:	:	FLUID	:	TEST	:	:	:	CaCo:	:								
2	1916	:	:	:	:	:	:	:	:	:								
:	Apr. 16	+	+	43	+	1	1	2	2	1	1	0	0	0	0	As.B.4	:	:
:	May 1	+	+	29	+	4	4	4	2	1	0	0	0	0	0	As.B.6	:	30
:	" 18	+	+	18	+	2	2	1	1	0	0	0	0	0	0	As.B.6	:	35
:	June 7	+	+	8	+	4	4	4	3	2	1	0	0	0	0	As.B.6	:	40
:	" 28	+	+	8	+	2	2	2	1	1	0	0	0	0	0	As.B.6	:	35
:	July 19	+	+	9	+	1	2	3	3	1	0	0	0	0	0	.9	:	40
:	Aug. 11	-	-	2	-	0	0	0	0	0	0	0	0	0	0	.9	:	40
:	Sept. 11	+	weak	8	+	1	1	1	1	1	0	0	0	0	0	.9	:	40
:	Oct. 23	+	+	4	+	2	2	3	3	1	1	0	0	0	0	.9	:	27
:	Dec. 23	+	+	5	+	1	1	2	2	1	0	0	0	0	0	.9	:	40
:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:
:	1917	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:
:	Feb. 16	+	+	7	+	1	2	2	1	0	0	0	0	0	0	.9	:	30
:	Apr. 14	+	+	1	+	1	2	2	1	1	0	0	0	0	0	.9	:	30
:	July 6	+	+	5	+	0	0	0	0	0	0	0	0	0	0	As.B.6	:	25
:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:
:	1919	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:
:	Jan. 27	-	-	6	-	0	1	1	1	0	0	0	0	0	0	As.B.6	:	:
:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:
:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:	:

Table II, Case II.—Taboparesis, with recovery from mental symptoms.

CASE III.—*Apparently early stage of paretic dementia. Marked mental and serologic improvement after one intravenous injection of neosalvarsan, followed by apparently complete arrest after intraspinal and inunction treatment.*

The patient, an unmarried business man fifty years old, was referred by Dr. J. A. Denney on July 8, 1915. He gave a history of "soft chancre" several times over thirty years ago. He had never been treated for syphilis. Prior to two years ago he had used considerable alcohol. Following an automobile accident in April, 1915, which resulted in fracture of the scapula and a scalp wound, he developed a nervous state. At the request of his associates, who noticed his decreased efficiency, he stayed away from work. He complained of a general lack of vigor. The pupils were slightly unequal and reacted very sluggishly to light. The tendon reflexes, especially the knee-jerks, were greatly increased. There was almost no tremor and his speech was normal. There was some decrease of pain sense on the arms. These signs, together with a positive Wassermann test on blood and spinal fluid, a positive globulin test, and a very strong gold reaction, led to a definite diagnosis of incipient paretic

dementia. It was, therefore, surprising that only two weeks after the first intravenous injection of neosalvarsan, 0.6 gm., the spinal fluid should be found practically normal and the blood Wassermann only weakly positive. After the second intraspinal treatment he returned to work, and since then he assures us that he has not felt so well for years. That this is not an abnormal feeling of well being is proved by the fact that he has done satisfactory work since August, 1915, and his nervous manner has disappeared.

In March, 1916, it was noted that his pupils reacted promptly, but the knee-jerks were considerably increased. In June the pupillary reaction again was more sluggish. However, his mental and general physical condition has remained excellent up to the present time. He has had mercurial inunctions most of the time since treatment was commenced.

CASE:	DATE	WASSERMANN	WASSERMANN	CELLS:NONNE-APELT:	LANGE	GOLD TEST	INTRAVENOUS	INTRASPINAL
:	:	BLOOD	SPINAL	GLOBULIN	:	:	NEOSALVARSAN:	SERUM
:	:	:	FLUID	TEST	:	:	:	O.C.:
3	1915	:	:	:	:	:	:	:
	July 8	+	+	10	+	2 5 5 5 4 3 3 1 0 0	.6	:
	" 22	+	-	5	+	1 1 1 0 0 0 0 0 0 0	.9	20
	Aug. 12	-	-	9	+	0 0 0 0 0 0 0 0 0 0	.9	25
	Oct. 1	+	-	4	-	1 1 0 0 0 0 0 0 0 0	.9	25
	Dec. 17	:	:	:	:	:	.9	:
	1916	:	:	:	:	:	:	:
	March 20	-	-	6	-	0 0 0 0 0 0 0 0 0 0	As.B.45	:
	June 21	+	weak	:	:	:	:	:
	July 5	:	+	3	-	0 0 0 0 0 0 0 0 0 0	.9	:
	" 17	:	:	:	:	:	.9	:
	Aug. 1	-	-	1	-	1 1 1 0 0 0 0 0 0 0	.9	30
	Oct. 30	-	-	0	weak	1 1 1 1 0 0 0 0 0 0	.9	:
	:	:	:	:	:	:	:	:
	:	:	:	:	:	:	:	:

Table III, Case III.—Arrest of disease.

The next patient has made an apparent mental recovery, but he paid a high price, having developed a spastic paraplegia, from which, after a lapse of two years, he only has sufficiently recovered to be able to walk a few steps unaided.

CASE IV.—A married business man, forty-two years old, was admitted to the hospital on December 13, 1916. He had been treated for secondary syphilis in 1908. Aside from being naturally irritable and complaining of gastric distress, he had apparently been well until three days prior to admittance, when he began to talk incoherently while on his way home from a business

trip to New York. When examined he was poorly oriented as to time, place and persons, and his memory for recent events was very poor. He had had his blood and spinal fluid tested the day before. The Wassermann test was strongly positive with both. The pupils were small, slightly unequal; the left one did not react to light, the right one only slightly; both reacted in accommodation. He had slight inco-ordination of the hands and feet, but the tendon reflexes were distinctly increased and there was apparently no sensory disturbances. Swift-Ellis treatments were given at rather short intervals (see table). Following the intraspinal injection of January 24th, he began to complain of numbness in both lower extremities and in the buttocks and genitalia. He was very unsteady, being unable to walk unaided. On January 27th he still could stand with his feet apart. The ataxia was greatly increased, but the strength in the legs was still fairly good. The tendon reflexes were brisk as before, and the cremasteric and abdominal reflexes were present, but he had developed a Babinski sign. The pain sense was diminished in the lower extremities, more so in the right thigh posteriorly, and on both buttocks. By this time his mental condition was considerably improved and continued to grow better rapidly. The legs, however, grew worse, and on February 10th a note was made that there was almost no movement at the right ankle and extension at the right knee was very weak. The left leg was less affected. On February 23d the following note was made: "Ankle movements about the same. Flexion of the thighs better. He cannot hold the legs up even when the knees are supported. Analgesia of both lower extremities and of buttocks and genitalia. Tactile sensation present. Left ankle-jerk absent, right normal. Knee-jerks increased, without clonus. Bilateral Babinski sign. Abdominal reflexes absent, cremasteric present."

We were here evidently dealing not with a meningeal but with a myelitic reaction. There was no stiffness of the neck and no pain in the head and back, but the rapidly progressing paralysis, ataxia, and analgesias of the lower extremities showed that a serious lesion of the cord substance, especially of the pos-

terior and lateral columns, existed. Fortunately, there was no incontinence of the sphincters, but micturition was rather slow. There was no unfavorable reaction from the subsequent intravenous injections nor from the small intraspinal injection on March 8th. From that time on the mental improvement was steady, and within a few months he became entirely normal mentally. The paraplegia, however, was exceedingly slow in improving. The left ankle-jerk, which had been absent, returned. The Babinski sign persisted. He learned to walk around the house supporting himself on heavy canes resting on pieces of wood about 6 inches square. It is noteworthy that the laboratory tests became practically negative. The mental condition has remained good and the patient now is able to take a few steps without help.

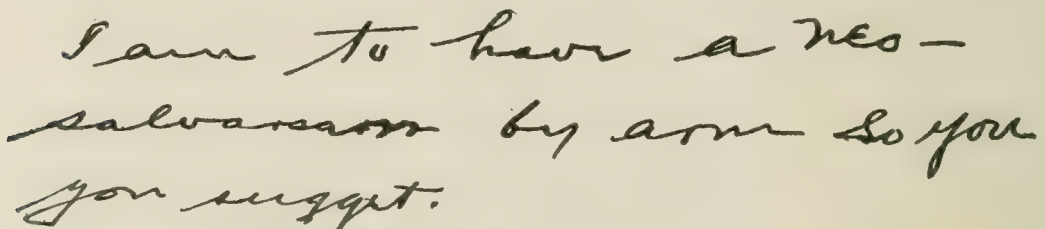
CASE:	DATE	WASSERMANN: BLOOD	WASSERMANN: SPIRAL FLUID	CELLS: NONNE-APELT: GLOBULIN TEST	LANGE GOLD TEST	INTRAVENOUS: NEOSALVARSAN	INTRASPINAL: SERUM
4	1916						
	Dec. 12	+	+	18		.6	
	" 19	+	+	20	1 2 4 4 4 3 1 1 0 0	.9	12
	" 28	+	+	10	3 4 4 4 3 2 1 0 0 0	.9	25
	1917						
	Jan. 3		+	8	1 1 2 2 0 0 0 0 0 0	.9	35
	" 12	+	+	8	2 3 3 3 2 1 0 0 0 0	.9	30
	" 24	+	+	3	1 1 2 2 1 0 0 0 0 0	.9	30
	Feb. 9					.9	
	March 2	+	+	7	3 4 5 5 5 3 1 1 0 0		
	" 8		+	4	0 1 2 2 1 1 0 0 0 0	.9	10
	April 7					.75	
	" 28					.75	
	May 16	-	-	3	3 2 2 2 3 1 0 0 0 0	.75	
	June 27					.75	
	Oct. 3	+	+	2	1 2 3 3 3 1 1 0 0 0	.9	
	" 13					.6	
	" 20					.6	
	Nov. 2					.6	
	" 16					.9	
	Dec. 5	-	-	2	1 1 1 1 1 0 0 0 0 0	.6	
	1918						
	Feb. 5	-	-	11	0 1 1 1 0 0 0 0 0 0	.8	
	March 15					.6	

Table IV, Case IV.—Arrest of mental symptoms. Myelitic reaction with paraplegia.

Of the cases which ultimately terminated fatally we will relate a few which possess interesting features. Thus, in the next case we had a prolonged remission during which the patient, a physician, was able to practice his profession. After thirty intraspinal injections he developed a spastic paraplegia and after prolonged invalidism he died.

CASE V.—*Mild but typical incipient paretic dementia, beginning in the autumn of 1913. Rapid improvement after commencement of treatment in March, 1914. Has had thirty intraspinal injections and almost continuous mercurial inunctions. Wassermann with spinal fluid remained positive, blood usually negative. Later spastic paraplegia and sphincter disturbance. Death in 1918.*

A physician thirty-seven years old, married, was first seen on March 17, 1914. No history of genital lesion, but about ten years ago he had a suspicious sore on a finger. He had been temperate in his habits, but had worked hard. In September, 1913, he became nervous, but improved after a brief rest. In December slurring of speech, tremor of the hands and lips, general irritability, and failing memory made their appearance. However, he was able to continue his work. On examination, his slurred speech, tremulousness, restlessness, and impatience at once suggested incipient paresis. Otherwise the physical findings were practically normal. The pupils were a little unequal, but reacted to light. As shown in the table, the Wassermann and other tests were strongly positive and confirmed the diagnosis. The patient was persuaded to take a rest. After receiving two Swift-Ellis treatments he went South for one month, and had one intravenous injection of neosalvarsan while there. The following fragment from a letter written on April 6th shows features of ominous significance in a man with a college education:



I am to have a neosalvarsan by am so you you suggest.

Fig. 23.—Specimen of patient's handwriting.

Improvement was steady and by the end of July his mental condition was nearly normal. After his return from the South late in April he lived in the country until September, coming to the hospital for his injections. He then resumed his work,

which he continued up to May, 1916. In addition to the injections mentioned in the table he has had inunctions of $\frac{1}{2}$ dram of mercurial ointment almost every day up to November, 1916, except for a few weeks in May and June. His wife has repeatedly noticed that he would become a little nervous three or four weeks after a treatment, and then immediately improve after the following one. A slight aggravation was also noted when the inunctions were discontinued for a short time. A brief relapse of mental confusion occurred in the beginning of March, 1915, when he had a slight indisposition with nausea, and one day of fever, the temperature reaching 102° F. Two days later he asserted that several men had entered his room, chloroformed him, dragged him to a house where many men with long noses and long nails pointed their fingers at him, and blew powder on his hair. At this time slurring of speech was again noticed for four days. When he came to the hospital for treatment two days after the dream episode he still insisted that it was a real experience, but he seemed otherwise perfectly clear.

Following this episode he engaged steadily in his profession, and it is not known that he has made any serious mistakes. There were no signs of any mental or physical aggravation of the disease at the time of the sudden rise of the cell count to 99 in February, 1916. When in the hospital for his injection on March 16th, however, he arose at 3 o'clock the following morning and insisted that it was time to go home. Late in April he was worse, confused at times, would frequently fall asleep during the day, once even at the breakfast table. When readmitted to the hospital on May 3d it is noteworthy that the blood Wassermann again became positive and the Lange test was unusually strong. After this treatment he grew still worse, did not know his wife at times, and in three days developed incontinence of urine. Two weeks later he had a fall, striking the lower abdomen, and then urinary retention set in. He could not walk unaided, and when examined on May 29th there was a distinct spastic weakness of both legs. The knee-jerks were increased and ankle-clonus and Babinski sign were present on both sides. Tactile sensation on the legs was normal. He soon improved mentally,

on the posterior surface of the thighs, outside of both legs below the knee, on glans penis and scrotum.

Later he again failed mentally and also grew weaker physically, becoming bedridden in November, 1917. He died on September 24, 1918. No necropsy.

Another fatal case of interest also is that of a physician who was rebellious to treatment, and finally developed a streptococcic meningitis of unusually long duration. Finally, there will be related a case of rapidly fatal pneumonia and meningitis following an intraspinal injection.

CASE VI.—*M., thirty-six years. Finger chancre in 1909. Attacks of headache, nausea, and numbness of right arm in 1913. Later right leg weak, mental failure. Slurred speech. Pupils unequal, left sluggish to light. Right Babinski and increased tendon reflexes. Four Swift-Ellis and two Ravaut injections without mental improvement. Developed purulent streptococcus meningitis lasting ten weeks, with peculiar symptoms; at first mental improvement and pupils again normal; fluctuations in spinal fluid condition, with interesting modifications of Lange test. Gradually failed and died. Brain examined postmortem.*

The patient, an unmarried physician, thirty-six years old, was referred by Dr. F. W. Allin on May 3, 1915. In 1909 he had a finger chancre, but no secondaries. No intemperance. In May, 1913, he developed pain in the right thigh, followed in October by attack of headache in left temple, nausea and vomiting for twenty-four hours, attended with numbness in the right arm, which lasted several days. In December he had a similar attack and later several others. Gradually the right leg became somewhat weak, so he walked with a cane. In the summer of 1914 he became irritable, emotional, and talkative, while he naturally was very reticent. He invested money foolishly, had extravagant ideas of wealth, and took unreasonable dislikes to people. In November, 1914, positive Wassermann test was obtained and he was given one injection of salvarsan, three of neosalvarsan, and considerable mercury. Slurring of speech had been noted for several months before his admittance

to the hospital. At this time the right pupil was wider than the left and reacted sluggishly to light. The left pupil reacted to light. The right knee- and ankle-jerks were increased and a Babinski sign was obtained on the right side. Considerable mental deterioration was manifest. Altogether the mental and physical signs and laboratory tests indicated a well-advanced parietic dementia. During the following two months there was no material mental change, but the right side seemed stronger. The table shows that an exceedingly strong Lange gold test was obtained on July 9th when neosalvarsan was injected directly into the spinal canal the first time, and that the fluid secured twelve days later showed a very weak Lange test and an almost negative Nonne-Apelt globulin test. We hoped for clinical improvement as well, but a most unfortunate complication arose.

After the second intraspinal injection of 3 mg. of neosalvarsan on July 21st the patient had a moderate reaction with headache, vomiting, and rise of temperature to 101.8° F. the next day. The next night he was delirious and he still had headache and vomited twice on the 24th. On the 25th he felt comfortable, had no fever, and was able to walk out of the hospital. The same evening headache returned and the temperature was 104° F. On July 26th the temperature reached 103° F., while the pulse-rate was 85. He complained of tinnitus, was very tremulous, and had involuntary urination and defecation. When visited at his home on July 27th he was unusually bright, although the temperature was 103.6° F. The neck was stiff, but there was no Kernig sign, and the tendon reflexes were practically normal. The optic disks were normal. Lumbar puncture yielded a cloudy fluid, but the pressure was not increased. There were over 3000 cells per cubic millimeter (see chart), nearly all polymorphonuclear. Diplococci, Gram-positive, were found in smears, and first thought to be pneumococci, but examination of the cultures by Dr. H. K. Nicoll showed them to be hemolytic streptococci. The patient also had a severe sore throat with white patches on the posterior pillars. Smears showed diplococci and streptococci.

On July 28th the patient returned to the hospital. He was somewhat restless, but unusually clear and sensible. The pupils now were equal and reacted much better to light than formerly. The neck was rigid. The Babinski sign had disappeared, but the tendon reflexes were a little stronger on the left side. The fever persisted, but aside from headache and restlessness the patient for several days maintained an improved mental condition and good physical strength. A lumbar puncture on July 31st, to our great surprise, yielded a clear fluid free from organisms both in smears and cultures, and with only 147 polymorphonuclear and 10 mononuclear cells per cubic millimeter. The modification of the Lange test after the onset of meningitis was quite interesting, showing on July 31st (see chart) the maximal reaction in the sixth tube, or dilution 1 : 320. The leukocyte count on July 28th was 10,000; August 1st, 9700; August 13th, 9350; August 19th, 14,600; September 2d, 10,600; September 10, 9600; September 19th, 9600; September 18th, 18,500.

Examination on August 5th: Patient has only slight headache. Still considerable stiffness of the neck, but only partial Kernig sign. Pupils equal and react to light. Optic disks normal. Knee-jerks brisk, more so on right side. Ankle-jerks about normal. Wrist- and elbow-jerks rather brisk, more so on right side. Slight right-sided facial weakness. During last forty-eight hours nearly constant twitchings in fingers of right hand. Plantar reflexes normal.

Severe chills occurred on August 13th and 19th. The twitchings in the right hand persisted; more or less headache was present most of the time. On September 7th moderate bilateral optic neuritis and slight external rectus weakness were noted. Kernig sign positive on both sides and Babinski sign on right side. September 27th neck was markedly rigid, head turned to left, right fist clenched. Coarse tremor and flexor contraction of right hand remain. Knee-jerks present, but ankle-jerks not obtained. Plantar reflexes normal. Marked emaciation. Pulse very weak. Hiccup present during last twenty-four hours. On the morning of October 4th he seemed to be

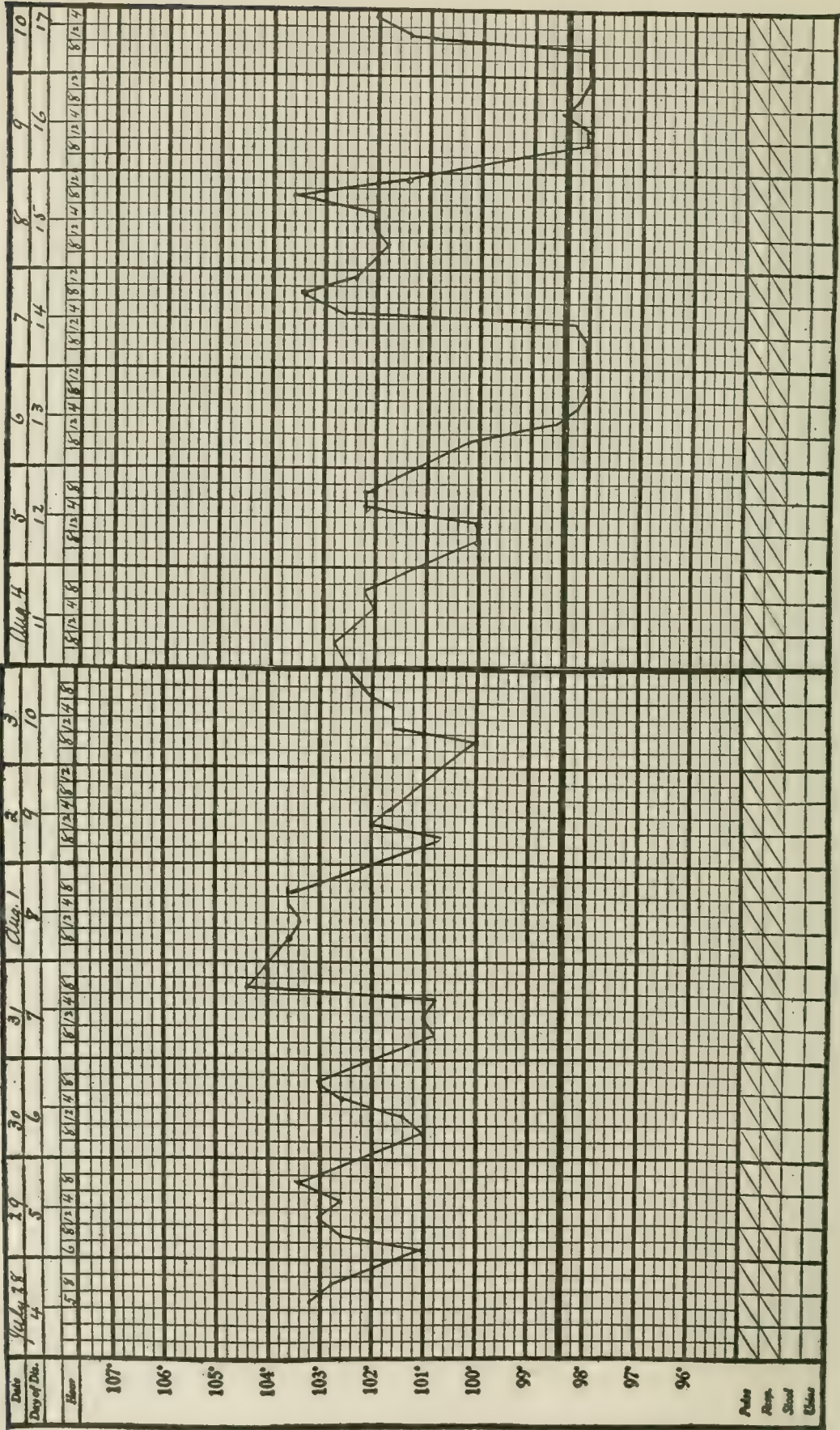


Fig. 24.—Case VI. Terminal streptococcus meningitis.

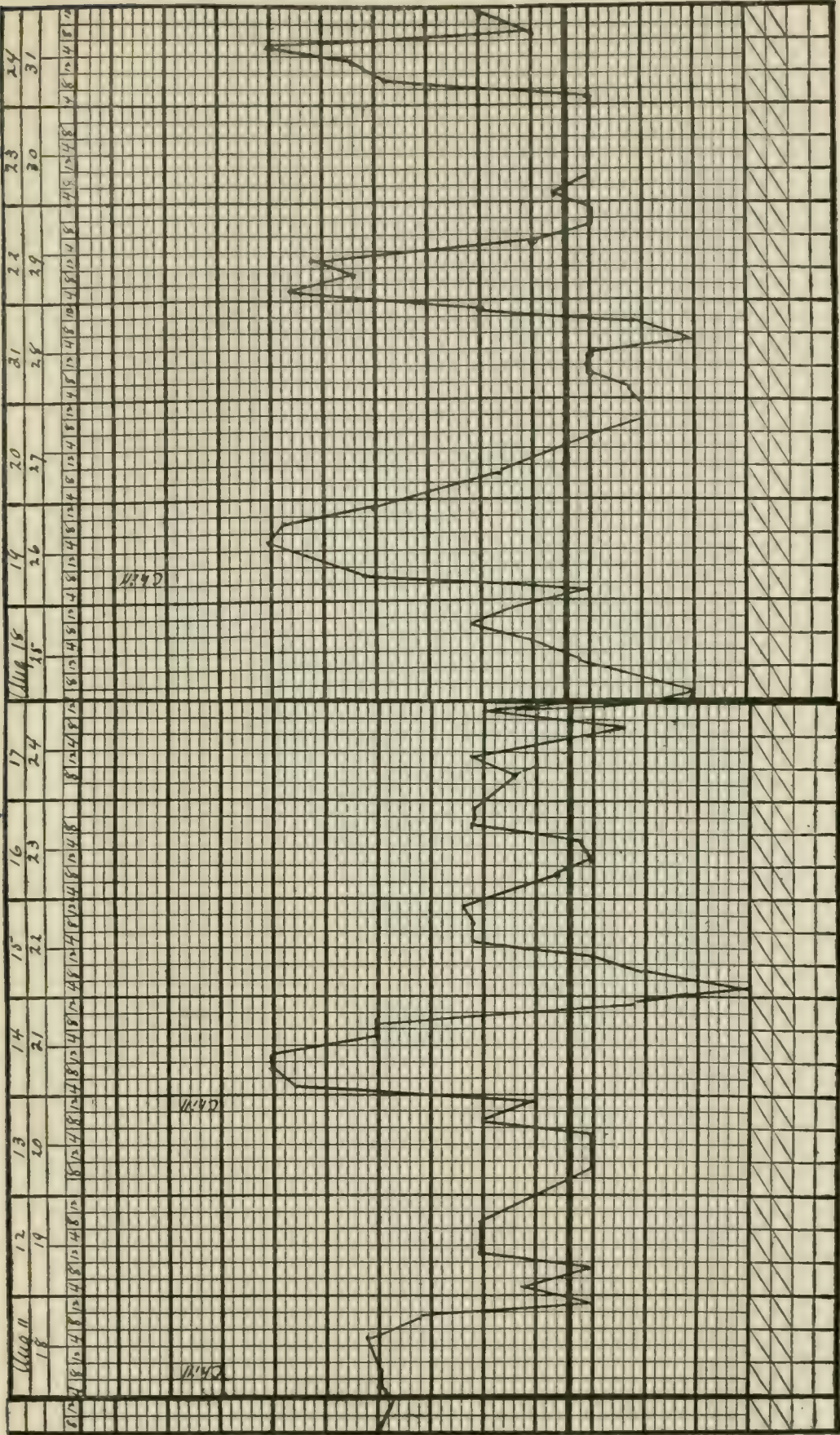


Fig. 25.—Fig. 24 continued.

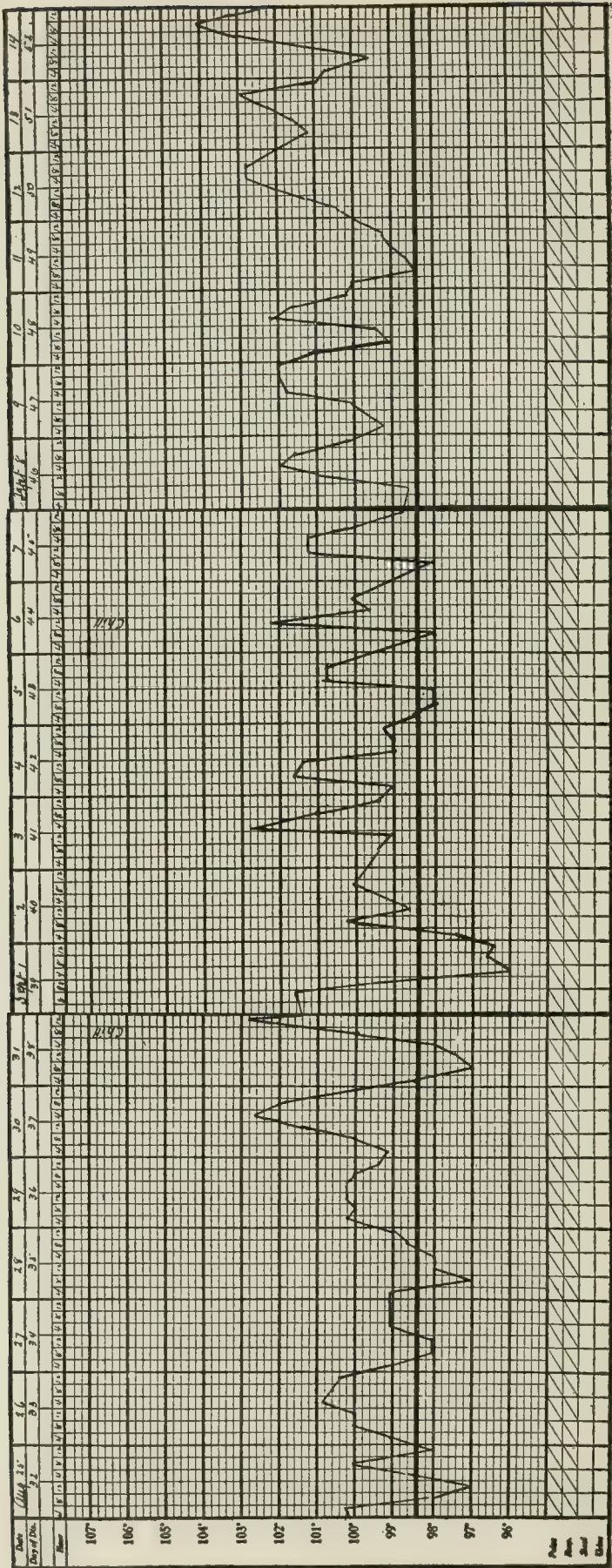


Fig. 26.—Fig. 24 continued.

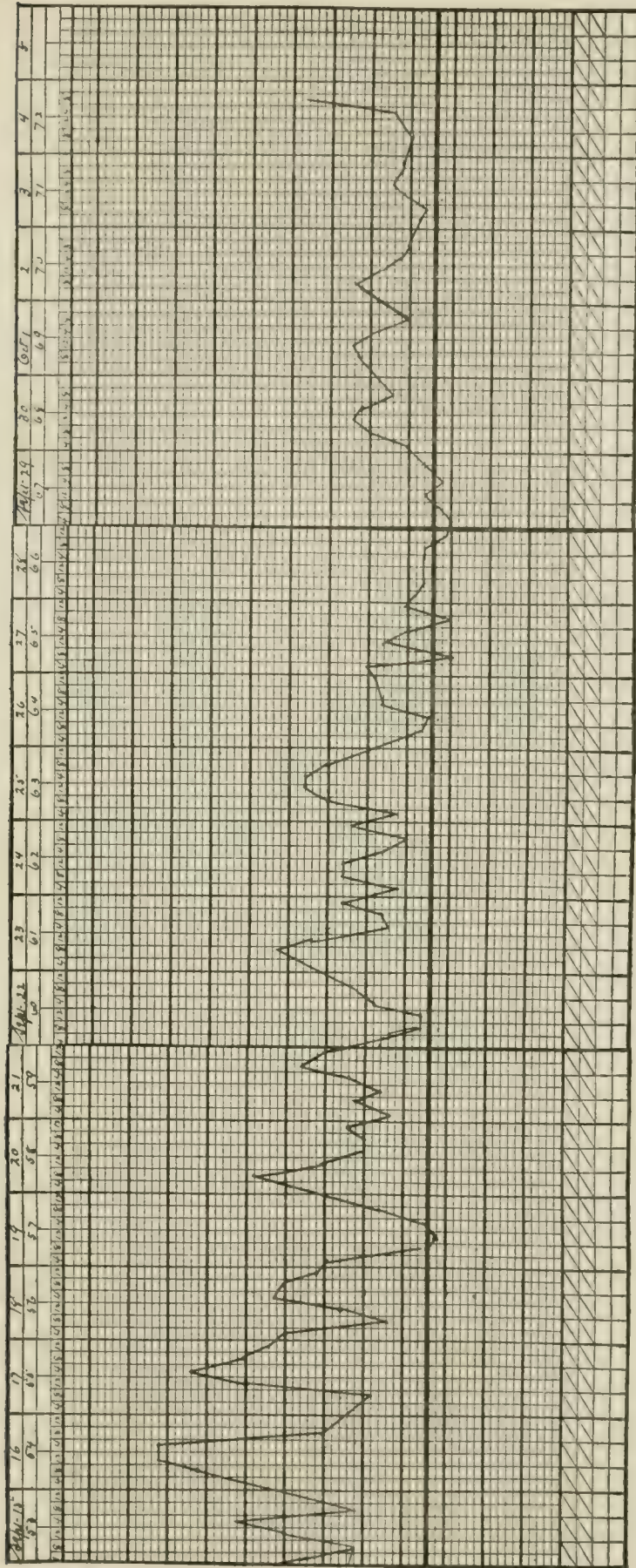


Fig. 27.—Fig. 24 concluded.

rational, but was unable to swallow. In the evening he became cyanotic and died.

The irregular fever is recorded on the accompanying temperature chart (Figs. 24-27).

The brain only was examined postmortem. It showed a fibrinous exudate, largely liquefied, on the pia covering the narrow, atropic convolutions. There was no abscess or other gross change on the cut surfaces. Histologic examination showed little evidence of acute inflammatory reaction. The pial meshwork was loose and edematous, with comparatively little perivascular inflammation, and most of the cells were mononuclear. The cortex also showed comparatively little perivascular infiltration. A section of the upper cervical cord showed distinct old degeneration in both columns of Goll and in both lateral pyramidal tracts, more marked on one side.

CASE:	DATE	WASSERMANN: BLOOD	WASSERMANN: SPINAL FLUID	CELLS: NONNE-APERT TEST	GLLOBULIN	LARGE GOLD TEST	INTRAVENOUS: NEOSALVARSAN	INTRASPINAL: SERUM :O.C.
6	1915							
	May 4	♦ ♦	♦	59	♦	5 5 4 4 4 1 0 0 0 0	.9	10
	" 14	♦ ♦	♦ ♦	17	♦	4 5 4 4 3 3 1 0 0 0	.9	30
	June 3	♦ ♦	♦	11	♦	3 3 4 3 3 0 0 0 0 0	.9	30
	" 18	♦ ♦	♦	7	♦	3 3 4 4 4 3 1 0 0 0	.9	25
	July 9		♦	8	♦	5 5 5 5 5 5 3 2 0	.9	2 mg.neos:
	" 21			4	♦ -	1 1 2 2 1 1 0 0 0 0	.9	3 mg.neos:
	" 27		♦ ♦	3052	♦ ♦	3 3 3 4 4 5 3 2 1 0	Fl.turbid	
	" 31			157	♦	2 2 3 3 4 5 2 1 0 0	Fl.clear -	Cultures neg:
	Aug. 2			409			Fl.cloudy -	" "
	" 20			433	♦ ♦	3 3 3 2 2 2 3 3 1 0	Fl.turbid -	no organism:
	Sept. 7		♦	1527	♦ ♦	3 5 5 5 5 4 4 3 2 1	Fl.turbid -	Cultures neg:

Table VI, Case VI.—Severe paretic dementia without response to treatment. Terminal septic meningitis.

CASE VII.—*Paretic man subject to epileptiform attacks. First two treatments uneventful. Twenty hours after third intraspinal injection of serum and 1 mg. neosalvarsan repeated convulsions, fever, signs of pneumonia. Spinal fluid purulent, teeming with pneumococci. Pneumococci in blood-culture. Death twelve hours after onset of convulsions.*

A machinist, thirty-three years old, was referred by Dr. Dean Lewis, to whose surgical service he had been admitted on account of epileptiform attacks which commenced in October, 1915. When admitted to the hospital on October 27, 1916, he presented distinct signs of paresis, such as slurred speech,

dementia, Argyll Robertson pupils, increased tendon reflexes. He improved after the first two treatments. On the morning after the third intraspinal injection he began having convulsions, became cyanotic, temperature rose rapidly to 105° F. (reached 107° F. before death), neck became rigid, and signs of consolidation of the right upper lobe appeared. The spinal fluid, which had been clear the day before, was now turbid, contained 10,000 polymorphonuclear cells per cubic millimeter, and enormous numbers of diplococci in smears. The puncture was made four hours after the onset of convulsions. Pure cultures of the pneumococcus were obtained from the spinal fluid and the blood. Death twelve hours after onset of convulsions. No necropsy.

CASE:	DATE	WASSERMANN : BLOOD	WASSERMANN : SPINAL FLUID	CELLS : GLOBULIN : TEST	NONNE-APERT : TEST	LANE GOLD TEST	INTRAVENOUS : NEOSALVARSAN :	INTRASPINAL : SERUM :
7	1916							
	Oct. 30	+	+	14	+	4 4 4 3 2 1 0 0 0		
	Nov. 3						.9	29
	" 14	+	+	17	+	5 5 5 3 3 2 2 1 1	.9	34+ 1 mg. neo:
	Dec. 6	-	+	5	+	5 5 5 5 3 1 0 0 0	.9	+
	" 7			10000			Fluid turbid	- Death.

Table VII.—Pneumococcus meningitis following intraspinal treatment.

CONCLUSIONS

1. A higher percentage of prolonged remissions, possibly also occasional arrest of the disease in paretic dementia is obtained with the combined Swift-Ellis and mercurial treatment than with simple intravenous salvarsan treatment, with or without mercury.

2. The treatment itself may occasionally lead to a temporary or permanent cord affection with sphincter disturbance and weakness of the legs, and may lower the resistance to acute infections with the pneumococcus or streptococcus.

3. Cases of taboparesis are relatively more favorable as to mental improvement, but the cord disease may be uninfluenced or grow worse.

4. The laboratory reactions at the beginning of the treatment furnish no prognostic signs as to its probable success or failure.

CLINIC OF DR. W. D. SANSUM

RUSH MEDICAL COLLEGE

THE TREATMENT OF CONSTIPATION

THE object of this clinic is to explain some of the more fundamental principles of anatomy, physiology, and hygiene that bear upon the subject of elimination by the bowel in as simple terms as possible.

Constipation is a condition in which the fecal material is retained for such a long time in the bowel that the stools become hard and dry because of the absorption of water. Every normal individual should have a daily movement of the bowels without the use of cathartics. This movement should be soft but formed, of the consistency of butter at average room temperature, and should equal about 6 ounces in weight, or nearly $\frac{1}{2}$ pint in bulk. A normal individual may have two or three movements per day provided the stools are formed.

Anatomy and Physiology.—Figure 28 represents somewhat diagrammatically the esophagus or gullet, the stomach, and the intestines, which together are called the alimentary tract.

Food taken by mouth passes down the esophagus to the stomach. The stomach is normally situated just beneath the diaphragm. In the adult it holds about 3 pints, but soon accommodates itself to the amount of food taken, so that it may hold only 1 pint or many pints. The digestive process begins in the stomach, but very little food is absorbed from the stomach. From the stomach the food passes into the small intestine.

The small intestine is a tube averaging about 27 feet in length. Here the digestive process is practically completed and nearly all of the food absorbed. From the small intestine the unabsorbed residue in a liquid form passes into the colon or large bowel.

The colon is a large tube averaging about $5\frac{1}{2}$ inches in diameter. It is about the size of one's forearm. The colon, as shown in Fig. 28, passes up the right side of the body, across beneath the stomach, up to the left of the stomach, and down

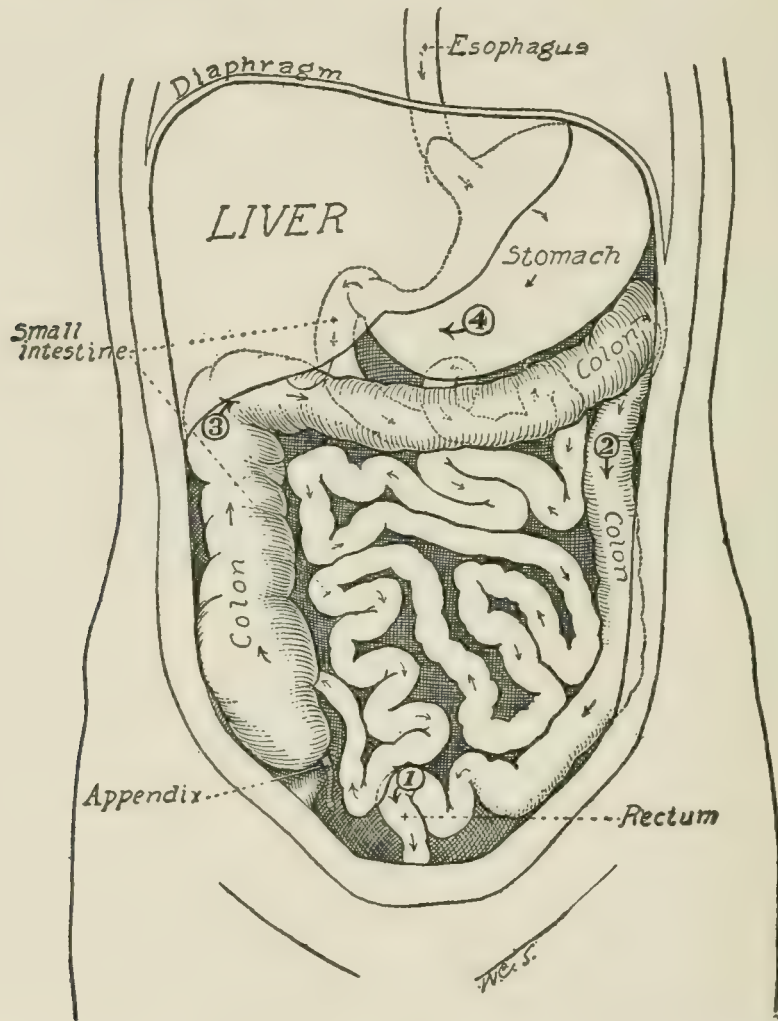


Fig. 28.—Diagram of the gastro-intestinal tract illustrating the rate of passage of food. Food entering the stomach (4) reaches the location (3) in the first twenty-four hours; the location (2) in the second twenty-four hours; and the location (1) the rectum, ready for extrusion, in the third twenty-four hours, taking three days to pass through the entire canal.

the left side of the body, where it terminates in the rectum. A knowledge of the location of the colon is important, since disturbances here are often wrongly diagnosed. Such disturbances may produce symptoms that resemble appendicitis, if the distress or pain is in the region of the appendix; or liver

trouble, if in the region of the liver; or stomach trouble, if in the region of the stomach. The indigestible residue less most of its water is forced on into the rectum in a semisolid state. In constipation the contents of the colon reach the rectum often in the form of small, hard, round masses.

The rectum is a very much smaller tube, about 5 inches in length, the function of which is to store the indigestible residues until they are expelled from the body. The rectum is closed at its lower end by the small sphincter muscles of the anal canal.

The contents of the alimentary tract is forced along by peristaltic waves. These waves are rather hard to describe, but you have seen them in freshly killed animals. Each one consists of an advancing constriction that passes from the stomach down, forcing a portion of the bowel content before it.

The bowel movement of today is in the rectum (Fig. 28, 1). The bowel movement of the next two days is in the colon (Fig. 28, 2, 3). The food taken on any single day furnishes the material for the bowel movement of the third or fourth day. On a large residue-containing diet the time may be shortened to forty-eight hours, but on a small residue-containing diet it often requires five days. This is one of the most important statements made in this discussion, and in talks with constipated patients seems to cause great surprise. It is a fact that is very easily demonstrated. Charcoal is indigestible and when passed makes a black stool. It therefore makes a good "marker." Patients are frequently asked to take a teaspoonful of powdered charcoal on the first day of treatment and watch for the black stool. With what we term an "anticonstipation diet," but which should be termed a "normal diet," the black stool usually appears on the third day after taking the charcoal.

Our bodies were planned a long time ago, when the alimentary tract was made especially large to accommodate the coarse foods eaten at that time. Civilization has changed our habits of eating. We demand the fine, white, patent flours from which nearly all of the residue has been removed. We live in large cities where the old-fashioned garden is an impossibility, and where, because of their perishable nature, cost of transpor-

tation, and low nutritive value, fresh vegetables and fruits are very expensive.

The chief bulk of the bowel movement comes from indigestible food residues. It is true that a part of the stool is made up of the dead and living bodies of bacteria which live in the colon and rectum especially, and that another part is made from secretions into the alimentary tract, but these portions are so small in amount that they may be safely ignored at this time. Let us, therefore, consider foods from the standpoint of their indigestible residues.

1. The grains (wheat, corn, etc.), prepared as they are today as fine white flours and breakfast cereals, have had most of their indigestible residues—as bran—removed. They are almost completely digested, leaving very little residue with which to make a bowel movement.

2. Meats, including fish and oysters, are almost completely digested and absorbed. Dogs fed wholly on meat have but one bowel movement in five days, and that is no larger than the bowel movement when on complete starvation. It is a very small black stool which consists chiefly of the secretion into the alimentary tract.

3. Eggs are almost completely digested and absorbed.

4. Milk and milk products, such as cream, cheese, butter, buttermilk, etc., are almost completely digested and absorbed.

5. Fats and oils of vegetable and animal origin are also almost completely digested and absorbed.

6. Sugars are another very widely used group of foods that leave no residue.

These six groups of foods together with the combinations made from them comprise the diet upon which most people like to live today. The result is inevitable. They may not have daily bowel movements without the use of cathartics, because the alimentary tract does not fill sufficiently.

Patients, therefore, resort to the use of cathartics. A drastic cathartic empties the whole alimentary tract, producing one or more watery stools. The patient is satisfied with the result. The next day he has no stool, but is not alarmed because he had

such a good movement the day before. The following day he has no stool, and he could not expect a stool even if he were on a diet containing a large amount of residue, as the colon has not yet had time to fill. He has been taught that a daily movement of the bowels is necessary for health, or at least he has had ample chance to learn from the advertising signs of patent medicine concerns. The active principle in nearly all of these patent medicines, from the herb teas to the pink liver pills, is some form of a cathartic. On the evening of the second day he invariably takes something for his bowels, and thus the cathartic habit is formed. Many persons take some form of a cathartic every other day, and many take something daily, thus never permitting the bowels to act naturally.

The continuous taking of cathartics soon leads to a serious condition of the colon known as colitis. The inner lining of the bowel is just as delicate as the inner lining of the eyelids. It contains no pain nerves. The pain nerves are in the outer coats of the bowel. No one would think of putting calomel, salts, castor oil, or cascara into the eye, because a red, painful, swollen eye would surely result. These things produce the same inflammatory condition of the lining of the bowel. The bowel "waters" in exactly the same way as the eye "waters." It is this "water" on a very large scale that produces the large watery bowel movement. Little flecks of a white substance would be seen in the corner of an eye so shamefully abused, and the same thing occurs in the bowel. This white substance, known as mucus, is seen in slimy masses in the stool. One often sees several ounces in a single stool from a person who has formed the cathartic habit.

The Treatment of Constipation.—With the above knowledge in mind, the treatment of constipation becomes very simple if too great damage has not been done. All one needs to do is to take a sufficient quantity of the foods that contain an indigestible residue and wait until the rectum fills up in the natural way.

The cell walls of plants are made up of a substance called cellulose. This is indigestible, and forms, with the water which it has the power of holding, the bulk of the normal stool. This

cellulose is found in the largest amounts in bran, vegetables, and fruits.

Bran, used either as a breakfast food or made into muffins, is our most valuable residue-producing substance. In fact, an ounce of dry bran with the water it absorbs makes several ounces of stool.

Bran contains mineral food in such quantities and of such value that a discussion of its food value is not out of place here. The farmer has known for a long time that stock thrive when bran is added to their diet, especially in the winter. Cows give more milk and their coats become smooth and shiny. This general improvement cannot be accounted for by the starch food value of the bran, for this is comparatively low.

The continuous cropping of the soil by wheat soon wears out the land, and we wonder why nature in her admirable economic plan permits such a thing to occur. A simple experiment in the germination of wheat graphically explains this. If from a number of kernels of wheat the bran is carefully removed, so as not to injure the wheat germs, and these are planted in a box with similar, but normal kernels, those with the bran removed slowly grow and are sickly looking plants, while those from the natural kernels are large and healthy. Nature has robbed the soil of the mineral foods that are essential to the growth of the baby plant and has placed them in a concentrated form in the bran of the wheat, thereby serving the double purpose of a protective covering for the wheat kernel and a storehouse of nourishment to be used by the baby plant until its root growth is sufficient for it to obtain this from the soil.

Vegetables form the next most important group of residue-producing foods. Spinach, cabbage, cauliflower, and carrots stand at the head of the list, producing a stool nearly equivalent to the amount of the vegetable taken. Potatoes produce about one-fourth their original bulk in stool. They are very valuable because they are palatable, rich in food value, and soothing to an irritable bowel. All vegetables are laxative in their effect.

Fruits form the next most important group of residue-producing foods. There is an old saying that "An apple a day

keeps the doctor away.” This is very true when we realize that at least one-half of the patients a physician is called upon to treat complain of constipation or some of its complications. Apple-sauce, like mashed potatoes, is palatable, rich in food value, and has a very soothing residue. Fruits in general cause a stool equal in amount to about one-fourth the bulk taken.

Many persons are able to keep their bowels moving daily by the generous use of fruits. Others find it necessary to use the coarser vegetables. The most obstinate cases need the addition of bran.

Occasionally bran, when used in large amounts, causes the stools to become mushy or even watery. In such cases it should be eliminated from the diet.

In the beginning of such a course of dietary treatment many patients complain of some discomfort. They claim that their stomachs do not hold more than 1 pint. They complain of a “stuffed” feeling. This is doubtless true, but by slowly increasing the amount of food both the stomach and large bowel gradually return to their normal state. If there is too much distress, omit the bran and instruct the patients to use larger amounts of cooked and finely divided fruits and vegetables. Mashed potatoes and apple sauce are very valuable at this stage of treatment.

Many patients become impatient and are unwilling to wait the necessary length of time for the bowels to act unaided, or it may be that a natural movement is prevented by the rectum being filled with impacted masses of feces. It is often wise during these first few days of treatment to use something that empties the rectum only. The oil retention enema is used for this purpose; 3 ounces of cottonseed oil are injected into the rectum and left there until the next morning, when it is easily expelled with the softened fecal matter. This is injected by means of a rectal tube and funnel similar to the one shown in Fig. 29.

The oil enema should be used at bedtime—

1. When there has been no stool during the entire day.
2. When the stool that day has been hard and dry.

If the oil enema has been neglected in the evening and there is no inclination to go to stool the next morning, a bowel movement should be obtained by using a small soap or glycerin suppository or the injection of from $\frac{1}{4}$ to $\frac{1}{2}$ pint of water. If this is not sufficient, a pint may be used, but a copious flushing must not be taken.

We find that about 2 pounds of fruits and vegetables together with a little bran is ample to produce from one to three natural movements per day. These amounts may soon be reduced.

The following cases under hospital management for constipation, among other ailments, illustrate the results of the application of the principles discussed above. All food given to these patients was carefully weighed for from six to eighteen days, and the stools resulting from such food carefully weighed.

Case No. 119,894, Presbyterian Hospital, Chicago:

The residue-producing substances in the diet amounted to 13 ounces of 5 per cent. vegetables and 4 ounces of bran made into bran muffins. The "charcoal marker" was given at noon of the first day. The black charcoal stool was passed on the morning of the third day. The patient had a daily soft-formed stool without the use of a cathartic. The black charcoal stool

together with the six that followed averaged 5.3 ounces each, and nearly half-filled the pint glass jars in which they were sent to the laboratory.

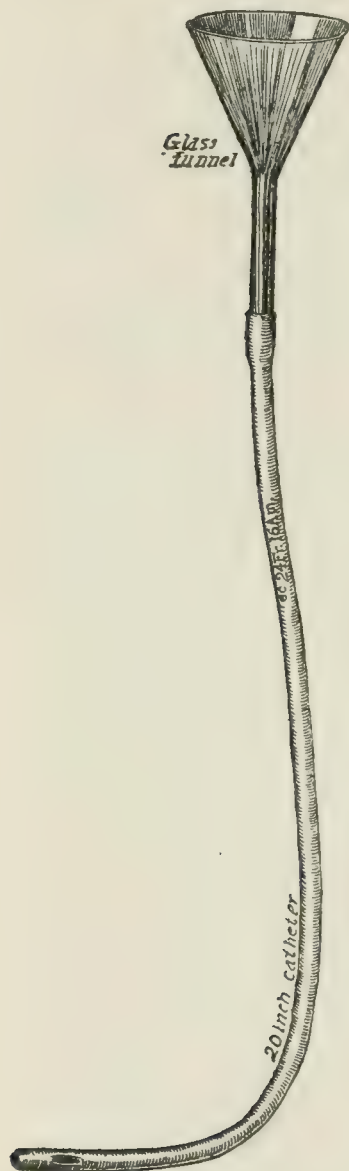


Fig. 29.—Funnel and catheter used for the injection of oil enemas.

Case No. 125,443, Presbyterian Hospital, Chicago:

The residue-producing substances in this diet amounted to 19 ounces of mixed fruits and vegetables and 2 ounces of bran made into bran muffins. The first stool appeared on the third day following the beginning of the diet. A charcoal marker given on the eighth day of treatment appeared in the stool on the tenth day. This diet produced a daily soft-formed stool, averaging 6 ounces in weight, over a period of eighteen days. These stools also nearly half-filled the pint glass jars in which they were sent to the laboratory.

A NORMAL DIET FOR MEN AND WOMEN ENGAGED IN ACTIVE BUSINESS OR HOUSEHOLD LIFE WHO WEIGH 125 POUNDS OR UNDER

This diet is called a normal diet. It is especially recommended to undernourished patients who are troubled with constipation.

It contains the food elements (carbohydrates, fats, proteins, and mineral foods) in the correct amounts and proportions to produce the maximum energy for the minimum amount of food. It is equivalent to 2800 calories or food units. It is ample in amount to produce a gain in weight of at least 1 pound per week. When the desired gain in weight is attained, or if no gain in weight is desired, the $\frac{1}{2}$ pint of cream may be omitted. It also contains enough of the residue-producing foods to insure at least one bowel movement per day without the use of cathartics. If the bowels move too freely the 5 per cent. vegetables may be reduced in amount or the bran muffins omitted.

The foods are arranged in two tables: one showing the total amounts recommended for consumption each day in ounces, interpreted in terms of common household measures; and a second showing how they may be apportioned between the three meals of the day. Other arrangements and combinations will suggest themselves. (Eggs, cream, butter, sugar, etc., may be made into pastries, ice-cream, or combined with vegetables.)

TABLE I

1. 8 oz. of 5 per cent. vegetables. One heaping tablespoonful of a cooked vegetable weighs about 1 ounce. (Lettuce, cucumbers, spinach, asparagus, rhubarb, endive, marrow, sorrel, sauer kraut, beet greens, swiss chard, celery, tomatoes, brussels sprouts, water cress, sea kale, cauliflower, egg plant, cabbage, radishes, leeks, string beans, broccoli.) Any vegetable may be eaten raw, cooked, made into salad, pickled, etc.

The percentage here refers to the amount of digestible starchy food which the vegetables contain.

2. 4 oz. of 10 to 15 per cent. vegetables. (Pumpkin, turnip, kolhrabi, squash, beets, carrots, onions, mushrooms, green peas, artichokes, parsnips, canned lima beans.)

3. 4 oz. 20 per cent. vegetables. (Potatoes, shell beans, baked beans, green corn.)

4. 4 oz. raw fruits in season. (Grape-fruit, lemons, oranges, cranberries, strawberries, blackberries, gooseberries, peaches, pineapples, watermelons, apples, pears, apricots, blueberries, cherries, currants, raspberries, huckleberries, plums, bananas, prunes, dates, figs.)

5. 2 oz. stewed or preserved fruits or honey.

6. 3 bran muffins.

7. 3 oz. bread. Six medium-sized slices.

8. 2 oz. butter. Six small cubes, as served in restaurants.

9. 8 oz. cream. One-half pint.

10. 3 oz. cooked cereal or 1 oz. of the dry special breakfast foods.

11. 2 oz. sugar.

12. 1 egg.

13. 5 oz. meat. Meat is usually bought by the pound and it is very easy to estimate ounce portions. (Fish, fowl, bacon, crab, cheese.)

14. 2 oz. pastry.

15. Tea, coffee, cocoa, chocolate, broth, Postum, pepper, salt, mustard, vinegar, etc., as desired.

TABLE II

Breakfast:

4 oz. raw fruit.

3 oz. cereal.

3 oz. cream.

1 oz. sugar.

1 oz. stewed fruit.

1 egg.

$\frac{1}{2}$ oz. bacon.

1 oz. bread.

1 bran muffin.

$\frac{2}{3}$ oz. butter.

Choice of some hot or cold drink.

Luncheon:

6 oz. soup.
2 oz. potato.
2 oz. 10 to 15 per cent. vegetables.
4 oz. 5 per cent. vegetables.
1 oz. bread.
1 bran muffin.
 $\frac{2}{3}$ oz. butter.
 $\frac{1}{2}$ oz. sugar.
2 oz. cream.
1 oz. stewed fruit.
1 oz. pastry.
Choice of some hot or cold drink.

Dinner:

6 oz. soup.
 $3\frac{1}{2}$ oz. meat.
2 oz. potato.
2 oz. 10 to 15 per cent. vegetables.
4 oz. 5 per cent. vegetables.
3 oz. cream.
 $\frac{1}{2}$ oz. sugar.
 $\frac{2}{3}$ oz. butter.
1 oz. bread.
1 bran muffin.
1 oz. pastry.
Choice of some hot or cold drink.

**A NORMAL DIET FOR MEN AND WOMEN ENGAGED IN ACTIVE
BUSINESS OR HOUSEHOLD LIFE WHO WEIGH BETWEEN
125 AND 150 POUNDS**

Within reasonable limits adults need food in proportion to their weight. In Table III the foods are similar to those of Tables I and II. Larger portions have been advised because of the added weight. The food value of this diet is about 3400 calories. It is ample to produce a gain in weight of about 1 pound per week. When the desired gain is attained or if no gain is desired, the cream may be omitted.

TABLE III

Breakfast:

4 oz. raw fruit (in season).
 4 oz. cooked cereal.
 3 oz. cream.
 1 oz. sugar.
 2 oz. stewed fruit.
 2 eggs.
 $\frac{1}{2}$ oz. bacon.
 1 oz. bread.
 1 bran muffin.
 $\frac{2}{3}$ oz. butter.
 6 oz. milk.
 Choice of some hot or cold drink.

Luncheon:

6 oz. soup.
 2 oz. potato.
 4 oz. 10 to 15 per cent. vegetable.
 1 oz. cold meat or cheese.
 4 oz. 5 per cent. vegetable.
 1 oz. bread.
 1 bran muffin.
 $\frac{2}{3}$ oz. butter.
 $\frac{1}{2}$ oz. sugar.
 2 oz. cream.
 6 oz. stewed fruit.
 1 oz. pastry.
 Choice of some hot or cold drink.

Dinner:

6 oz. soup.
 $3\frac{1}{2}$ oz. meat.
 2 oz. potato.
 4 oz. 10 to 15 per cent. vegetables.
 4 oz. 5 per cent. vegetables.
 3 oz. cream.
 4 oz. milk.
 $\frac{1}{2}$ oz. sugar.
 $\frac{2}{3}$ oz. butter.
 1 oz. bread.
 1 oz. bran muffin.
 1 oz. pastry.
 Choice of some hot or cold drink.

Bran Muffins

2 cups bran.
 1 teaspoonful soda.
 3 tablespoonfuls molasses.

1 cup white flour.
 $\frac{1}{4}$ cup butter.
 $1\frac{1}{2}$ cups of sour milk.

Mix dry ingredients. Rub in the butter with the tips of the fingers. Add molasses, sour milk, and soda. Put in hot muffin tins and bake in hot oven twenty to thirty minutes. Quantity sufficient for 9 muffins.

Bran Muffins

2 cups bran.	1 cup white flour.
3 tablespoonfuls of brown sugar.	$\frac{1}{4}$ cup molasses.
1 cup sweet milk.	1 teaspoonful soda.
2 tablespoonfuls melted butter.	1 teaspoonful salt.
1 beaten egg.	$\frac{3}{4}$ cup raisins.

(These may be omitted.)

Mix egg, butter, sugar, salt, and milk. Add soda dissolved in molasses. Then add bran and flour. Bake in moderate oven. Quantity sufficient for 12 muffins.

Agar-agar is a preparation made from seaweed. It is a very pure form of cellulose. It is, therefore, indigestible. A small amount of dry agar-agar absorbs water and holds it very firmly, so that 1 ounce of the dry agar-agar makes several ounces of stool. It is perhaps the least harmful of all of the popular substances used by constipated patients. It is not palatable, has no food value, and is expensive. Properly instructed patients prefer to take their residue in the form of fruits and vegetables.

Mineral oil is one of the by-products obtained from petroleum. It is sometimes called liquid vaselin. It is not absorbed from the alimentary tract. In the language of this clinic this is an indigestible residue—1 ounce of mineral oil produces 1 ounce of stool. Many of these oils contain irritating impurities and their continued use causes an irritation of the colon. Even if they were pure and tasteless, properly instructed patients would prefer their indigestible residue in the form of apple-sauce, etc.

Prunes, Figs, and Dates.—These fruits contain laxative substances similar in nature to cascara, and if used in sufficient quantities to produce loose, watery stools may be as harmful as the taking of mild cathartics. Used in moderate amounts they seem to do no harm. The cathartic effect of the so-called “Fig Syrups” is due to senna or some other active cathartic element.

Cathartics Following Surgical Operations.—Following surgical operations, especially upon the alimentary tract, it is often unwise to fill the bowel with masses of indigestible residue. Laxatives are, therefore, used to keep the bowels moving properly. Such patients often get the idea that this is the proper way to keep the bowels moving, and continue the practice long after they leave the hospital.

Miscellaneous.—There are a number of procedures, such as the various forms of abdominal massage and the drinking of large amounts of hot or cold water before breakfast when the stomach is empty, which stimulate peristalsis. If the bowel is not properly filled with residue material either these methods fail to produce the desired bowel movement or they must be carried to such an extreme that they are harmful. The various forms of massage are time consuming and unnecessary if the proper diet is taken.

CLINIC OF DR. GEORGE F. DICK

ST. JOSEPH'S HOSPITAL

AN UNUSUAL CASE OF TYPHOID FEVER

THIS case of typhoid fever is of interest because of the difficulty it presented in diagnosis.

The case is that of a white woman, aged forty-seven years; married; without children; and with no history of previous disease. Her health was good until she made a trip to Florida. Four weeks before admission to the hospital she had an attack of severe diarrhea. At the same time she had headache and muscular soreness, but no nausea or vomiting. After a few days her condition was enough improved to allow her to return to Chicago. A week later the diarrhea recurred, with but slight constitutional symptoms. At the end of another week she complained of feeling "feverish"; the diarrhea continued and the patient went to bed and called in a physician. After a week's care in bed she was sent to the hospital. On admission she had recovered from the diarrhea and her only complaint was a feeling of weakness with a tendency to sweating. Her temperature was 102° F.

Physical examination showed a well-nourished woman in good general condition. The tongue was coated. In the skin of the chest and abdomen there were about half a dozen rose spots. There was no general adenopathy. Nothing abnormal was found on examination of the chest. There was slight generalized abdominal tenderness, with some tympanites without rigidity. The abdominal wall was thick and the spleen was not palpable, but the area of splenic dulness was somewhat increased. As shown in the chart, the pulse-rate was low in relation to the temperature. The leukocyte count was 6000 (Figs. 30-33).

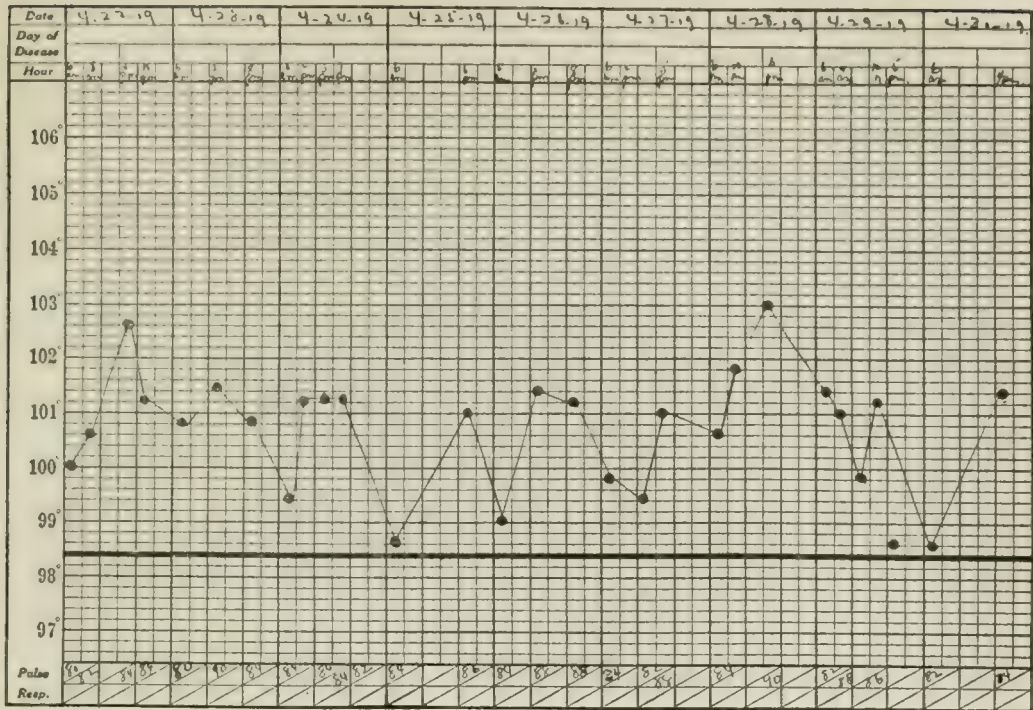
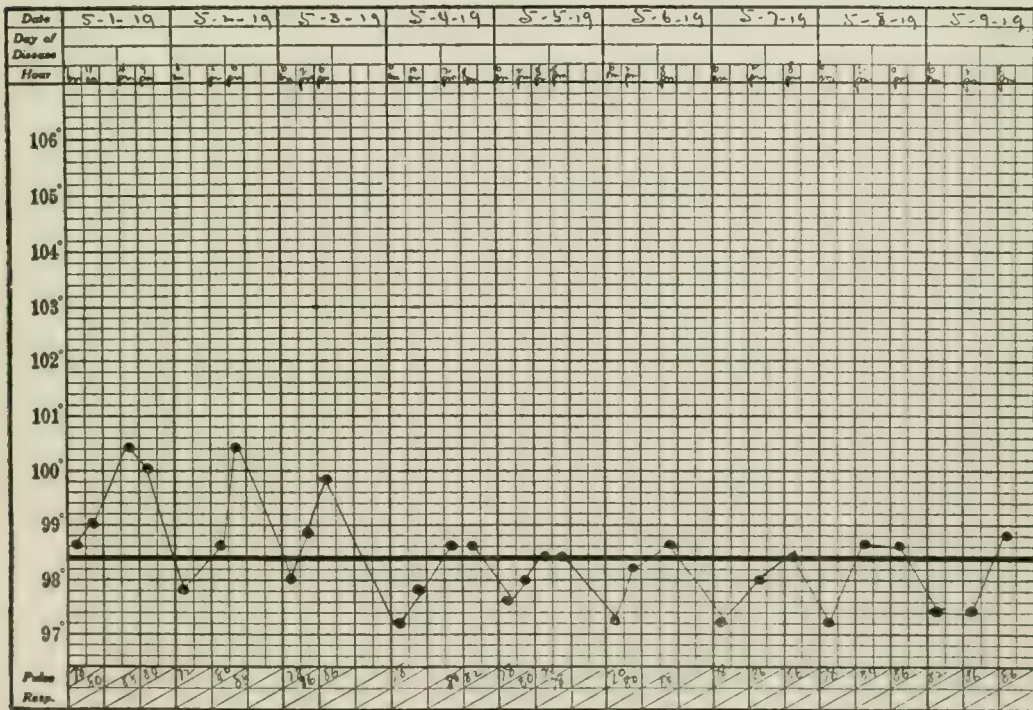


Fig. 32.



Since the patient had been in bed with fever for a week, it was thought that the probable diagnosis of typhoid fever would be confirmed by a Widal test. The patient's serum, however, caused no clumping or loss of motility in three strains of typhoid vacilli; one of which was the Rawling's strain.

A blood-culture made at this time was negative, and cultures of the urine and feces on lactose-litmus agar plates failed to yield any colonies of the typhoid group of organisms.

Because the clinical findings indicated that this was a typical case of mild typhoid or paratyphoid fever, the cultures from the urine and feces were repeated at intervals of three or four days, and agglutination tests were made with *Bacillus typhosus* and *Bacillus paratyphosus* A and B at the same intervals. All these cultures and tests were negative.

The temperature for four weeks was irregular, ranging from 98° to 102° F. During the fifth week it rose to 103° F. and pus appeared in the urine. Simultaneously there appeared in the cultures of the urine and feces numerous colonies which did not ferment lactose. Further study showed that these colonies contained Gram-negative bacilli which, while they were definitely motile, were much less so than the usual typhoid bacilli.

The bacillus isolated fermented mannite and maltose, producing acid without gas. It did not ferment lactose or saccharose and did not produce indol. Compared with other strains of typhoid, it fermented dextrose but feebly.

Agglutination tests were made during the sixth week of the disease and after the acute infection of the urinary tract, using the patient's serum with three strains of known typhoid bacilli, paratyphoid bacilli A and B, and the organism isolated from the patient's urine and feces. The organism isolated from the patient's urine and feces was agglutinated by the patient's serum in serum dilution of 1 : 640. Paratyphoid A was agglutinated in a serum dilution of 1 : 80. Paratyphoid B and the known typhoid strains were not agglutinated.

The temperature became normal and the pus disappeared from the urine following the administration of hexamethylenamin

in 15-grain doses; an amount sufficient to give a test for formaldehyd in this patient's urine.

Summary.—The case may be summarized as one clinically typical of mild typhoid fever with a complicating acute urinary infection. It is unusual in that the Widal test was persistently negative with all strains of the typhoid bacillus tested except the strain isolated from the patient.

The organism obtained in cultures of the urine and feces is identified by its morphology and its biochemic action as a typhoid bacillus, but differs from the known strains of typhoid bacilli used as controls in the agglutination tests, and in its weak fermentation of dextrose.

It is possible that other cases of typhoid fever with persistently negative Widal reactions might show an agglutination with their own strain of typhoid bacillus.

CLINIC OF DR. JAMES G. CARR

MERCY HOSPITAL

CARDIAC ARHYTHMIAS

THE man whom we present to you today as our first patient is thirty-seven years of age, English by birth, a bricklayer by trade, who has, however, followed the occupation of automobile mechanic for some time. He was first seen at Northwestern Dispensary in March, 1918. At that time he complained of a more or less constant irregularity of the pulse, varied by attacks of shortness of breath, with a sense of thoracic constriction, some nausea and occasional vomiting, and a very rapid, regular pulse. Upon examination, aside from the irregularity of the heart-beat, no cardiac abnormality was noted. A Wassermann, + + + +, was present. Syphilis was denied and the patient attributed his cardiac trouble to typhoid fever, which he had in 1905. He stated very positively that since his typhoid he had noticed irregularity of the pulse. He was put on specific treatment without result. A later Wassermann was negative. The evidence does not warrant the assumption of a specific basis for the cardiac irregularity; rather is it probable that we are dealing with a condition which is definitely related to the above-mentioned attack of typhoid fever.

Though the patient was seen at the dispensary from time to time, we were unable to examine him in one of the attacks of tachycardia of which he still complains. These come on abruptly and usually terminate just as quickly. The longest time that an attack has lasted was about nine hours; during the past winter it happens that the attacks have been infrequent.

On palpating the radial pulse we find a frequent small beat occurring soon after the preceding beat, and followed, after a

longer interval than the normal one between two beats, by a strong beat; occasionally there is a long pause corresponding in time to the interval of time covered by two normal beats. We find in listening over the heart that at these times of missed radial beat the ventricle has contracted early without transmitting an impulse to the wrist; this is sufficient evidence that we are not dealing with a partial heart-block. Tracing of the radial pulse shows the irregularity very clearly; there is, however, a "dominant rhythm." The distance from the up-stroke of the normal beat preceding the weak beat to the up-stroke of the beat succeeding the weak beat is equal to the distance from the up-stroke of a normal beat to the up-stroke of the second succeeding normal beat. In other words, the weak beat and the succeeding long pause, or the time between two beats when we

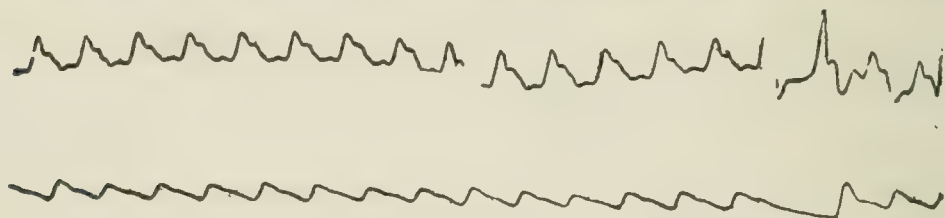


Fig. 34.—Case I. Premature ventricular contraction.

detect an intermediate ventricular contraction missed at the wrist, is equal to the time occupied by two normal beats. There is a distinct compensatory pause following each abnormal ventricular contraction. Furthermore, the beat succeeding the long pause is stronger than the normal; still further, the patient's statement that his pulse is regular as it becomes more rapid is significant. We have here the three cardinal signs of premature ventricular contraction: first, a compensatory pause following the premature beat (where the beat is missed at the wrist, the long pause between the preceding and succeeding beats is equal to the time occupied by two normal beats); second, the first beat following the compensatory pause is especially strong; third, as the pulse quickens the irregularity diminishes or disappears (Fig. 34).

Premature ventricular contractions are not the result of a single cause, nor are they associated with any particular type

of heart disease. Experimentally we know little about their origin except that electric stimulation may excite them, and distention of either ventricle by artificially raising arterial pressure may also produce these abnormal ventricular contractions. Clinically they occur in several large groups of cases: (1) Infections—the case here presented is one of premature ventricular contractions occurring probably as the result of myocardial change resulting from typhoid; other infections, especially syphilis, may be responsible. (2) Toxic—tobacco and coffee may be the etiologic factors; the toxic type of irregularity may be associated with exophthalmic goiter. (3) Overwork—the so-called “athlete’s heart” may show this form of irregularity. (4) Hypertension. (5) Arteriosclerosis. (6) Various forms of valvular disease. (7) Neurasthenic conditions. (8) Finally, a large number of people over forty show an occasional dropped beat more or less constantly, for which a satisfactory explanation cannot be offered.

From another standpoint we may classify the etiologic factors as:

- (1) Organic—associated with myocardial change, about which we can say little precisely:

Infections.

Hypertrophy of overwork.

Hypertension.

Arteriosclerosis.

Valvular disease.

Exophthalmic goiter.

- (2) Functional:

Toxic—tobacco, coffee, exophthalmic goiter. Neurasthenia.

- (3) A large group in which we have no warrant for assuming disease.

The proximate cause of this type of irregularity is doubtless an abnormal irritability of the ventricular musculature, as a result of which an early ventricular contraction occurs. This contraction is weak for two reasons: first, because the usual diastolic period has been cut short and the ventricle has not had

time to fill properly; second, because the ventricle has contracted early before it has regained its full contractile power. The auricular impulses continue regular, but the impulse coming from the auricle immediately following the premature ventricular contraction finds the ventricle in the refractory stage, unable to respond; thus the compensatory pause is due to the fact that the ventricle does not again contract until the ensuing auricular impulse comes through; now the ventricle, well filled during the long pause, and having regained its full contractile power, contracts with unusual force.

Clinically, the diagnosis of premature ventricular contraction must be made from partial heart-block and auricular fibrillation. This can usually be done without the aid of instruments. Where heart-block is present the missed beat at the wrist is associated with an entire absence of ventricular contraction during the same period; in premature ventricular contraction with missed beats at the wrist the early contraction can be felt or heard at the apex. Auricular fibrillation has a perpetual irregularity; there is no "dominant rhythm." The beats are absolutely unequal and irregular and rhythm is not to be thought of in connection with them. Strong beats may follow short pauses, weak beats occur after long pauses, and the irregularity is much worse as the pulse quickens.

Treatment in these cases is mainly directed to the etiologic factor, where this can be discovered. In cases due to a definite toxic agent, as tobacco and coffee, the indication for treatment is clear. In general, the principle of disregarding the irregularity, so far as definite medicinal treatment is concerned, and treating the underlying cause is good. Cardiac stimulants, especially strychnin, digitalis, and caffein, are not indicated; they are more likely to do harm than to help, and should be withheld.

The diagnosis of premature ventricular contractions from auricular fibrillation is of importance because of the relationship of these conditions to the use of digitalis. Premature ventricular contractions in themselves never indicate the use of digitalis; on the contrary, most cases of this irregularity will not do well under digitalis; the drug will frequently increase the irritability

of the cardiac muscle, sometimes making the irregularity worse, and, therefore, is usually contraindicated in premature ventricular contractions. Where this type of irregularity is present with broken compensation, digitalis should be used for the decompensation; the irregularity is ignored. On the other hand, auricular fibrillation is the type of cardiac irregularity in which digitalis is of the most service. Nothing gives more brilliant results than digitalis in cases of decompensation with fibrillation, and many cases are kept on small doses of the drug with benefit after compensation is restored. It is of the utmost importance to differentiate the pulse of the "regular irregularity" from that of the absolute and perpetual irregularity.

In some cases of premature ventricular contraction on a nervous basis bromid will be of service. In general, these patients may be assured that the irregularity is of no significance and may be ignored.



Fig. 35.—Case II. Premature ventricular contraction, with subsequent pulsus alternans.

The second patient is fifty-eight years of age, Irish born, an employee of the hospital, who was admitted to the ward on account of a "cold"; on admission his temperature was 100° F. This promptly subsided and his acute infection has been of no moment. He has a well-marked emphysema; arteriosclerosis is prominent; the radials are hard and easily rolled under the finger. Blood-pressure is 155 systolic, 100 diastolic. A twenty-four-hour specimen of urine amounted to 750 c.c.; contained no albumin or sugar; specific gravity was 1014; a phthalein test showed 60 per cent. in two hours. There is present a cardiac irregularity of the type already described. Here the etiologic factor is doubtless arteriosclerosis. The arrhythmia in itself calls for no treatment; it is probably an incidence of myocardial change secondary to arteriosclerosis, but we cannot make the heart beat regularly with drugs. There is no indication for the use of digitalis (Fig. 35).

The third patient has been under our care in Northwestern Dispensary for broken compensation. He is sixty-five years old, a laborer by occupation. At present the signs of decompensation are not prominent; the apex-beat is in the fifth interspace $10\frac{1}{2}$ cm. to the left of the midsternal line; his blood-pressure is systolic 224, diastolic 110. The urine shows much albumin and many hyaline and granular casts; the specific gravity is 1015; phthalein excretion in two hours is 22 per cent. He presents, in addition, the type of cardiac irregularity we have been discussing. The tracing (Fig. 36) shows premature ventricular contractions with the compensatory pause followed by the strong beat. We also see the evidence of many missed beats; the premature beats do not always appear at the wrist; we have discussed the necessity of making a distinction from heart-block under such circumstances; the stethoscope reveals premature ventricular contraction during the long pause at the wrist.

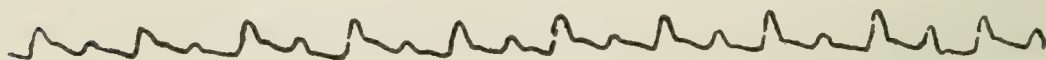


Fig. 36.—Case III. Pulsus alternans.

This man has been given digitalis not because of his arrhythmia, but in spite of it; his decompensation is much less marked. The premature ventricular contraction is not an indication for digitalis, but in connection with signs and symptoms of decompensation it is not a contraindication. The cardiac patient who has no evidence of decompensation should not have digitalis, except the cases in which a decompensation once under control can only be kept so by the continued administration of small doses of the drug, with occasional periods in which it is omitted.

The tracing further shows a variation in the height of the pulse-waves; a strong beat is followed regularly by a weak one. This alternation of strong and weak beats is the condition known as pulsus alternans. In this particular case you will note that the alternation is most marked immediately after the occurrence of premature ventricular contractions. Dr. Paul White, of Boston, has called our attention to the fact that the earliest manifestation of the tendency to permanent pulsus alternans may be

the appearance of the alternation after premature ventricular contraction and at no other time.

Pulsus alternans, in all probability, means a badly damaged myocardium. It is of special importance in prognosis. In a series of cases Paul White found a mortality of 100 per cent. within three years in his cases of permanent pulsus alternans. The cases in which the alternation occurred after premature ventricular contractions are not so unfavorable. The explanation commonly offered for this condition is as follows: Following the larger beat (the normal one in which the contractile power of the cardiac muscle is exhausted by the effort of contracting) the interval until the succeeding beat is not sufficient for the muscle fully to regain the power of contractility; hence, the succeeding beat is weak; after another interval the heart is recovered sufficiently to produce a normal contraction.

The recognition of pulsus alternans is possible not only by means of the radial tracing, but often by ordinary palpation of the radial. In this particular case our attention was first called to the alternation when we were estimating the blood-pressure; it was discovered that above a pressure of 200 the pulse-rate was only half of that when the pressure was less than 200; and about that particular point the beats as they came through were quite distinctly alternately strong and weak; the strong beats produced a sound up to a pressure of 224.

Pulsus alternans must be differentiated from the dicrotic pulse and the *pulsus bigeminus*, the pulse of coupled beats. The diagnosis from the dicrotic pulse rests on the failure to find a cardiac beat corresponding to the dicrotic impulse at the wrist. *Pulsus bigeminus*, the coupled beat, occurs especially as the result of too much digitalis, and we may say in passing that the occurrence of the pulsus bigeminus in the course of the administration of digitalis is an absolute indication for at least a temporary discontinuance of the drug. In *pulsus bigeminus* the second beat is nearer to the preceding than to the succeeding beat; there is a long pause following the weak beat; there is a regular premature ventricular contraction occurring early after the normal ventricular contraction. In *pulsus alternans* the beats

are equidistant; the time interval between the contractions is the same between the strong and the succeeding weak beat, between the weak and the succeeding strong beat; or, again, the weak beats are midway between the strong beats. At least, the weak beat is never early; it does not approach the preceding strong beat; if it varies from a midposition, and if the interval between the beats is not regular, it is likely to be the result of the deficient contractile power of the heart; hence, the weak beat may be beyond the midpoint and nearer to the succeeding strong beat.

The differentiation between *pulsus bigeminus* and *pulsus alternans* is of the utmost importance from the standpoint of treatment. *Pulsus bigeminus* may be regarded as a contraindication for the use of digitalis. On the other hand, digitalis may be used freely in the presence of *pulsus alternans* associated with other indications for the drug. A rather recent article by Windle has emphasized the value of digitalis in cases showing *pulsus alternans*. In this particular case a misinterpretation of the type of arrhythmia present would be serious; to diagnose the bigeminal pulse and withhold digitalis would be to keep from this patient the one drug which offers the most help.

The next patient is a man forty-four years of age, a dining-car waiter, whom we first saw in the County Hospital two years ago; there he was a patient for fifteen weeks. He was then suffering with broken compensation from which he recovered slowly. He has a mitral regurgitation with a marked dilatation of the heart, but at present his compensation is good. Four years ago he had inflammatory rheumatism; at sixteen he had syphilis; his present cardiac trouble can probably be attributed to his rheumatism, as he has shown two negative Wassermann reactions in the past two years and presents no stigmata of syphilis. In June, 1917, we made a radial tracing which showed a *pulsus alternans*. Since that time this alternation has been constantly present. A recent tracing shows a typical and exceptionally well-marked alternation; yet this patient is better today than he has been for some time. Some months after his discharge from the County Hospital he returned to his work in the dining-

car. He has worked steadily for about eighteen months, and has felt quite well in spite of this well-defined alternation. During much of this time he has taken digitalis in small doses; during the past winter, however, he has taken or omitted the drug largely on his own initiative. The case is of especial interest on account of the patient's generally good condition so long after the demonstration of an alternation.

Briefly to review the cases as they have been presented, we have seen 4 cases presenting some abnormalities of cardiac rhythm; the first a case of premature ventricular contractions with a history of paroxysmal tachycardia, the whole having developed after an attack of typhoid, may properly be ascribed to myocardial change subsequent to that infection. The second, a case of the same type of arrhythmia on an arteriosclerotic basis; the third, another case of premature ventricular contraction in association in this instance with hypertension, showed also the pulsus alternans, especially marked after the premature contraction; and the fourth, a characteristic case of pulsus alternans occurring in the course of mitral disease.

A CASE OF SYPHILITIC PERIOSTITIS OF THE HUMERUS

THE patient, a colored man forty-eight years of age, a laborer in the stockyards, came to the dispensary on account of inability to use his right arm. For several days in the latter part of March he had some pain in the arm; since April 1st he has been unable to work. He came under our observation April 11th; at that time, as it present, his chief complaint was of pain on attempting to lift the arm above the level of the shoulder; there is no pain while the arm hangs at the side. The patient does not definitely localize the pain; he places it in the joint or somewhat below it.

There is no redness or swelling of the shoulder-joint; the joint is not tender; there are no growths about the joint, no crepitus on motion, no impairment of mobility except when an effort is made to raise the arm above the level of the shoulder. There is no tenderness along the course of the nerves. On resistance, we find the power of the arm well maintained; the reflexes are normal. There is no atrophy or edema of the arm, and the other joints are entirely free from symptoms. The axillary glands are not enlarged, there is no palpable tumor above the clavicle or in the axilla; there is no evidence of cervical rib, which is an occasional cause of pain in the arm.

The one positive finding in addition to the inability to raise the arm is tenderness over the inner aspect of the humerus, localized over a point covered by the tips of two fingers, about 2 inches below the head of the humerus; pressure at this point invariably elicits evidence of pain. It appears that this is really the point of greatest pain when the attempt is made to raise the arm. The symptoms then may be attributed to disease outside the joint; neither the pain nor tenderness is confined to the course of any nerve, nor have we the severe pain usually found as a symptom of neuritis; and the pain and tenderness are localized. We feel that we can eliminate neuritis and myositis as well as arthritis. The symptoms are not those of an acute osteomyelitis;



Fig. 37.—Dr. Carr's case of syphilitic periostitis of humerus—right.

we can feel no irregularity in the bone suggestive of tumor; there is no history of tuberculosis elsewhere, nor are there any findings



Fig. 38.—Dr. Carr's case of syphilitic periostitis of humerus—normal left.

locally suggestive of tuberculosis. There is no history of syphilis, but the patient admits several attacks of gonorrhea. We

regarded the case as a probable periostitis of syphilitic origin; the pain on raising the arm is probably due to tension on the muscles attached to the inner border of the humerus. The roentgenogram confirms the diagnosis of periostitis (Figs. 37, 38); on comparing the pictures of the two shoulders we can see the thickness and irregularity of the periosteum along the inner border of the right humerus, near the upper end and extending almost into the joint. The Wassermann reaction is strongly positive. We believe we are correct in regarding this as a syphilitic periostitis.

The chief interest in this case lies in the fact that syphilitic periostitis is not common in the humerus; the tibia, the cranial bones, the clavicle, less often the ribs, are the common sites of this manifestation of specific disease. There is no evidence of involvement of the joint either on physical examination or in the roentgenogram. The patient has already been given mercurial ointment for inunction and is taking iodid of potassium. We will begin injections of some one of the arsphenamin preparations shortly.

A PULMONARY ABSCESS FOLLOWING TONSILLECTOMY

THE patient to whom we now invite your attention was in the hospital as a patient from April 10th to April 29th one year ago. He was then twenty-nine years of age. He is American born, one of our own medical students. Ten days before his admission to the hospital last year his tonsils were removed. The operation was done under gas and ether. Six days after the operation he had a pain in the upper part of the right chest, aggravated by cough and deep breathing; four days later, while walking on the street, he was taken with a severe fit of coughing, during which he brought up a large amount of sputum; that evening he was admitted to the hospital and his temperature on admission was 104° F.

Physical examination disclosed an area of impaired resonance immediately below the right clavicle, and over this area the voice and breath sounds were exaggerated, but were not distinctly tubular; tactile fremitus was increased and moist râles were audible. The temperature on the morning of the 11th was 100.2° F. and the leukocyte count was 10,400; the general condition was good, with slight cough and expectoration. On the 13th the temperature rose to 103.2° F., and with the rise of temperature the patient felt badly, had no appetite, and was quite prostrated; during the night of that day he coughed a great deal, bringing up large quantities of sputum, which, on examination, showed three distinct layers, the lower one a thick and heavy sediment, the middle thin and serous, the upper frothy; the whole being of a dark greenish color. Elastic fibers were present, but tubercle bacilli were not found. After this exacerbation the patient was better, but twice within the next week this experience was repeated. Each time the temperature went up, and the patient, without being told of the temperature rise, expressed himself as feeling badly; he anticipated the cough and expectoration which brought the expected relief. After the

expectoration in these two instances the percussion note over the area below the right clavicle already described became distinctly hyperresonant; this hyperresonance gradually disappeared, manifestly as the secretion accumulated. The roentgenogram shows definite change in the area described (Fig. 39).

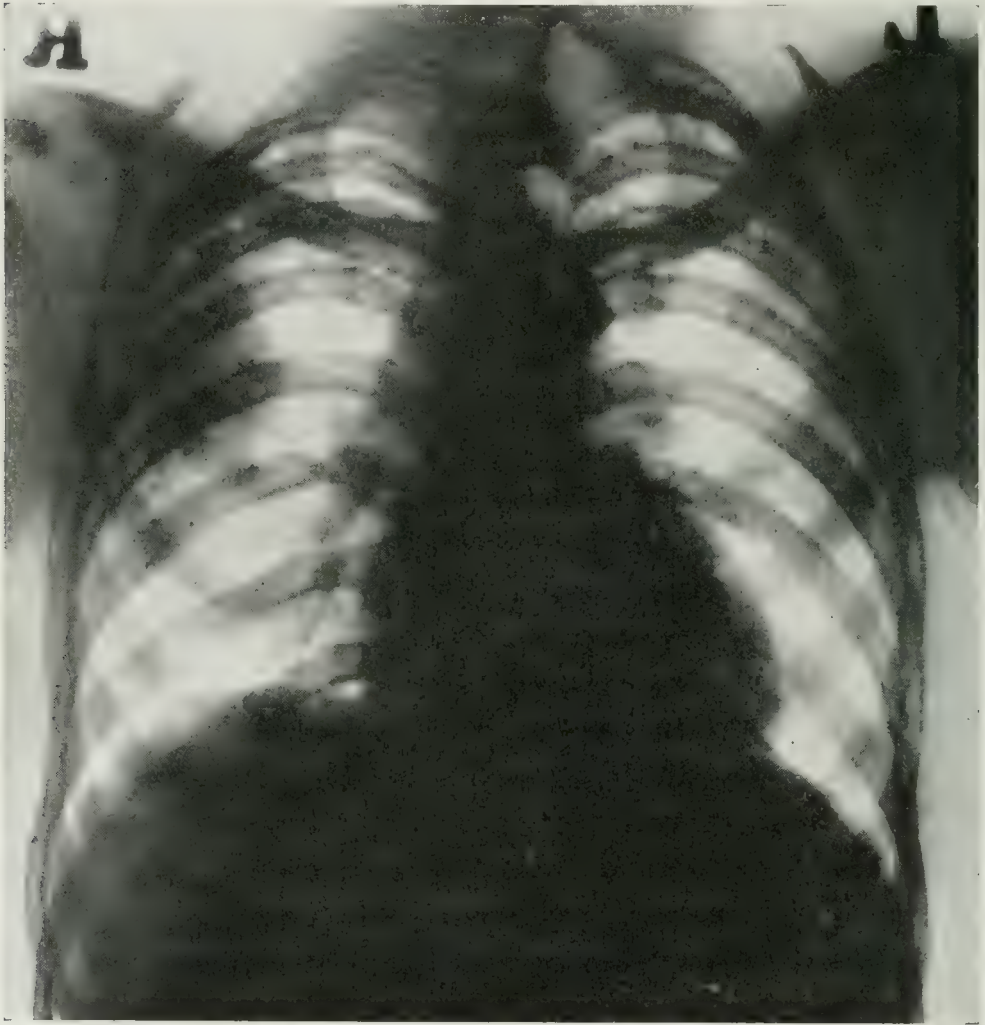


Fig. 39.—Dr. Carr's case of lung abscess. Right side.

After the 20th of the month the temperature remained low, at times touching normal, and at his own urgent request the patient was allowed to return to his home in Utah. The treatment outlined for him was that usually ordered for tuberculosis—fresh air, plenty of food and rest in bed, until the return of the temperature to normal. For a time he improved steadily,

but in July, possibly because of impatience on his part and of too much activity, he became worse, and late in that month had to undergo an operation for empyema, at which time portions of the eighth and ninth ribs below the lower angle of the right scapula were resected. After an extremely stormy course he made a slow recovery, returning to Chicago in February of this

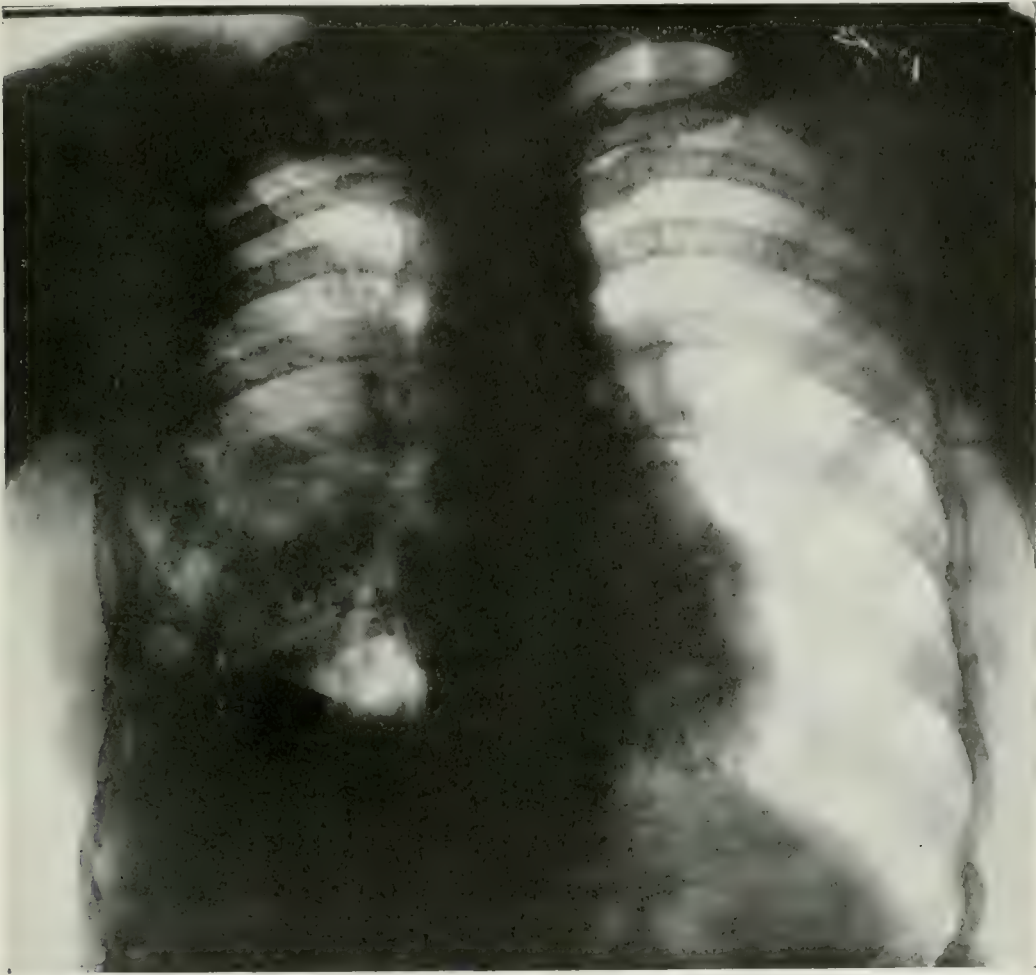


Fig. 40.—Dr. Carr's case of lung abscess one year later, showing fibrosis of the lung and pleural thickening.

year to resume his school work. He still had a discharging sinus, which has been closed now for about three weeks. At present, aside from being under weight, his general condition is good; he has no fever, no cough except a slight cough early in the morning, with no characteristic expectoration; the appetite is good and the patient feels very well.

On physical examination the right chest anteriorly over the area described as the seat of the original process shows only very slight change, but posteriorly below the lower angle of the scapula and in the axilla there is dulness, with diminished fremitus, voice and breath sounds, and respiratory excursion is absent. Râles are not heard. The roentgenogram shows a shadow, which probably represents a mass of adhesions rather than fluid, as the diaphragmatic angle is not obliterated; the lung shows a diffuse fibrosis (Fig. 40). The active process has subsided, and the patient may be regarded as having recovered, though with considerable diminution of pulmonary function, as the result of permanent changes in the right lung and pleural cavity.

Etiologically, this case is related to the tonsillectomy performed six days before the onset of pain in the right side, and ten days before the fit of coughing with the discharge of pus. Other cases of a similar etiology have been reported; Manges, of New York, reported 9 such cases some two years ago; the case is not unique, which is all the more reason for calling your attention to it. *Too often the possible dangers in connection with tonsillectomy are overlooked; there are dangers connected with this operation, and the mere presence of moderately enlarged tonsils is not an indication for tonsillectomy. The operation should be performed in the presence of definite indications, with due regard to the possibility of serious results.*

In general, lung abscess occurs after pneumonia of either type; aspiration pneumonia, especially of the insane, is often the cause, and foreign bodies, trauma, septic and ulcerative conditions of the upper respiratory tract, embolism, and rupture of contiguous accumulations of pus may be causative factors. It is only in recent years that our attention has been called to the occurrence of abscess of the lung following tonsillectomy.

The important points in diagnosis are: (1) Constitutional symptoms of an infection (in old cases these may be subordinate), (2) the character of the sputum, especially the expectoration of large quantities at varying intervals of time, and the presence of elastic tissue fibers, (3) an etiologic factor, (4) physical findings of localized pulmonary infiltration, (5) radiographic find-

ings, (6) leukocytosis. In this particular case the leukocyte count was of no significance.

The treatment which we employed was purely expectant—rest in bed and careful forcing of the feeding, with provision for fresh air. In many cases creosote may be employed to advantage. In regard to the matter of operation and of treatment in general, our own opinions may be expressed in the words of Dr. Lord, of Boston, "The indications for or against operation are difficult to formulate, and each case must be decided on its merits, but, as a general rule, it may be stated concerning the acute cases that in the presence of a small process without marked symptoms of sepsis, with purulent and not foul expectoration and without a large amount of elastic tissue and lung shreds, an expectant policy may be followed. If after observation from three to four weeks recovery or marked improvement does not occur, operation should be considered. Operation is indicated, on the other hand, with an extensive process, marked sepsis, putrid sputum, and abundant elastic tissue or lung shreds. The exigencies of the individual case determine the propriety of operative interference in cases which have lasted for months or years. The condition may be intolerable to the patient or so menacing to life as to justify surgical interference, even though little more than partial relief may be expected. Circumscribed, inextensive, and single lesions offer a greater hope of success."

CLINIC OF DR. ARTHUR F. BYFIELD

COOK COUNTY HOSPITAL

SOME ASPECTS OF HODGKIN'S DISEASE

My plan is to discuss with you certain phases of Hodgkin's disease from material furnished by a case under observation for nearly a year and which has recently gone to autopsy. The diagnosis, made probable during life by the history, clinical course, and physical and laboratory findings, was made positive by the microscopic characteristics of an excised lymph-node. Further verification, were it needed, has been given by the autopsy data.

Inasmuch as we have already on several occasions discussed the disease as a clinical whole, we intend at this time to deviate from the usual plan of presentation by limiting ourselves to a discussion of selected aspects of the condition on the basis of data furnished by this particular case.

Nomenclature.—It is essential, first of all, to grasp clearly the present-day interpretation of the term "Hodgkin's disease." Infectious, or malignant, granuloma is descriptive of the nature of the morbid process, but is, more or less, an inclusive term, as there are other infectious granulomata, and other granulomas may have a malignant tendency, though much less often, it is true, than is the case with Hodgkin's disease. Used in the strict sense which we are about to define, *Hodgkin's disease* should, by all means, be the name employed.

There are still a great many physicians, unfortunately, to whom the term Hodgkin's disease suggests a complex of conditions characterized essentially by a progressive enlargement of the lymphatic nodes, beginning usually in the cervical region, and pursuing a malignant course. They do not appreciate that the

only absolute criterion of the process is the histopathology; for, on the basis of the latter alone, are we enabled to exclude those other conditions—tuberculous and syphilitic enlargements of the lymph-nodes, lymphoid leukemia, lymphosarcoma, metastatic carcinoma of the nodes, and still other processes—which were included in Hodgkin's original description of the disease bearing his name.

The use of the term *pseudoleukemia* to denote the poorly defined group of diseases included in Hodgkin's monograph, or as synonymous with Hodgkin's disease in the strict sense, must also be condemned. It is unfortunate that many of our textbooks continue to use the two terms interchangeably. The resemblance of Hodgkin's disease to leukemia—lymphatic leukemia—is a superficial one, and doubt as to which of the two conditions is present should last only until a gland has been excised and microscopically examined. There is a sharp difference in the pathologic histology of the two processes, a difference which serves to exclude definitely from the leukemias (specifically, lymphatic aleukemias) those cases of malignant granuloma in which confusion is most likely to arise, *i. e.*, Hodgkin's disease with little or no increase in the total white cell count, but with a considerable lymphocytosis. (Well-marked leukocytoses in Hodgkin's disease are generally of the polynuclear variety, allowing ready differentiation from the usual form of lymphatic leukemia.)

Pseudoleukemia is not Hodgkin's disease, but is a genuine lymphatic leukemia with characteristic leukemic histologic changes in the lymphoid tissues and exhibiting a relative—and perhaps absolute—increase in the lymphocytes with a normal or subnormal total white cell count (aleukemic leukemia).

Hodgkin's disease, we repeat, is the best available name for that infectious granuloma, of malignant tendency, which we are considering. The diagnosis rests ultimately upon a specific histologic picture which is quite different from that of other conditions sometimes included under the same caption. And, conversely, pseudoleukemia as a synonym for Hodgkin's disease had best be dropped from our nomenclature, as it is a misnomer

in the light of our present knowledge of Hodgkin's disease on the one hand, and of the several types of lymphoid leukemia on the other.

The Onset.—*Cutaneous Manifestations.*—In the great majority of cases the disease begins insidiously with a progressive, painless enlargement of certain lymph-nodes. In some 10 per cent. of the cases, however—and ours is one of this category—there occurs early in the course a very significant cutaneous manifestation, namely, pruritus. Our patient stated that he began to experience spells of almost intolerable itching soon after he noticed the enlarged lymph-nodes in the neck, and that the resulting scratching produced small areas of bleeding all over the body. These skin changes, together with small crusts and spots of pigment, were present when he first came under observation.

Somewhat later there appeared discrete, slightly raised, rose-colored, urticaria-like lesions over the upper half of the body, together with a moderate number of split-pea-sized, papular, necrotic lesions of general distribution. The last were of the nature of prurigo.

The cutaneous manifestations of Hodgkin's disease may accompany the earliest lymph-node enlargements, or they may precede them by some months; while in a few cases they occur a variable time after the establishment of the disease. In some instances pruritus is the only skin manifestation; the excoriations and pigmentation are secondary, due to the scratching. In the case under consideration the itching was symptomatic, apparently, of no cutaneous lesion, and appeared simultaneously with the cervical tumors. In some cases a prurigo-like exanthem occurs, a condition observed fairly late in our patient. The urticarial lesions are probably part of the prurigo picture. A wide-spread eczematous condition is another itching dermatosis sometimes observed.

These various itching lesions may be fairly constant, or they may come and go with the onset and subsidence, respectively, of recurrent febrile periods, or they may follow the excision of a lymph-node. As distinguished from the lesions about to be des-

cribed, they are more or less evanescent in character and are due, possibly, to the toxin generated by the specific infectious agent.

Quite different from the lesions just described are the tumor-like growths in the skin and the deep, ulcerative processes which are occasionally observed. These are true cutaneous manifestations of the disease proper, as is evidenced by the fact that on excision and examination they show the specific histologic picture of the disease.

The second prodromal symptom in point of frequency, namely, diarrhea, did not occur in our case.

The Temperature.—As is the case with many—if not most—of the signs and symptoms of Hodgkin's disease, the temperature curve varies in different cases and in the same case at different periods. In the case under discussion the temperature for the greater part of the time was of the continuous type, for short periods high—103° or 104° F.—but more often not exceeding 101° F. Afebrile periods were also observed.

This is perhaps the commonest form of febrile reaction in the disease, in which there is generally, at one time or another, fever of some type. A recurrent fever is, however, the most striking type observed; and the association of periods of high temperature (with the usual daily variations) and a renewed activity on the part of the lymph-nodes—either in the form of an enlargement of those already involved or of fresh involvement—has led to the use by some of the name *recurrent glandular fever* to describe the disease.

Another febrile type occasionally encountered is the remittent, the curve of which is very similar to that of a quotidian malarial or pyemic infection.

The matter of the temperature has been taken up for consideration because an attempted analysis of its cause leads us into the interesting field of the specific mycotic factor in the disease. Hodgkin's disease is unquestionably of infectious origin, and it cannot at present be denied that any or all of the different febrile types described may be due to the toxins of the specific bacterial agent. However, it also cannot be denied that in the two more striking febrile forms—the recurrent and

the remittent—the possibility of a secondary pyogenic infection is strongly suggested. This view is strengthened by the fact that it is not uncommon during periods of activity in the disease to observe high polynuclear leukocytoses, and also that nodes removed at such times are likely to be characterized microscopically by marked polynuclear infiltration. We are all familiar, furthermore, with the findings, in some cases, of streptococci in excised nodes and in the circulating blood.

We have no intention, at this time, of entering in detail into the matter of recent studies on the specific relation of the diphtheroid bacillus (*Corynebacterium granulomatis maligni*) or of the streptococcus to Hodgkin's disease.¹ Suffice it to say that the former is almost certainly not the specific microbic agent and that the streptococcus plays only a secondary rôle in the process. We shall return to this point in our discussion of the autopsy findings.

The Blood.—The following are the blood findings in tabular form:

Count No.	Hemo- globin.	Red cells.	White cells.	Lympho- cytes, per cent.	Large monos., per cent.	Poly. neu- trophils, per cent.	Poly. eosino- phils, per cent.	Poly. baso- phils, per cent.
I	85	5,056,000	33,000	8.0	.04	88.0	0.0	0.0
II	70	4,232,000	36,800	7.0	0.0	88.0	0.0	5.0
III	69	4,832,000	35,000	11.5	5.0	82.0	1.25	0.25
IV	90	5,224,000	41,600	11.0	3.0	85.0	0.0	0.0
V	78	4,160,000	24,800	1.0	1.0	98.0	0.0	0.0
VI	78	4,800,000	41,200	14.0	1.0	85.0	0.0	0.0
VII	82	4,656,000	33,400	15.0	3.0	82.0	0.0	0.0
VIII	82	4,320,000	31,600	9.0	0.0	91.0	0.0	0.0
IX	70	4,000,000	30,000	9.0	1.0	90.0	0.0	0.0
X	33,600	16.0	2.0	82.0	0.0	0.0
XI	24,250	21.0	1.0	78.0	0.0	0.0
XII	34,000	14.0	1.0	84.0	1.0	0.0
XIII	54	34,200					
XIV	55	34,300					
XV	61	3,110,000	28,800	13.0	1.0	86.0	0.0	0.0
XVI	26,000	7.0	3.0	89.0		1.0
XVII	32,100	13.0	2.0	84.0	1.0	0.0
XVIII	60	3,000,000	30,000	16.0	2.0	82.0	0.0	0.0
XIX			33,600	21.0	1.0	78.0	0.0	0.0

¹ The reader is referred to an article by us, covering this subject, in the *Amer. Jour. Med. Sci.*, 1918, clv, 409.

The hemoglobin-red blood-cell ratio throughout was that of a secondary anemia, which is almost without exception the case in the disease. The erythrocytes exhibited practically no variations from the normal. The blood-platelets, as judged from the stained specimen, were moderately increased in number.

The behavior of the leukocytes in Hodgkin's disease is always of particular interest, due in large measure, perhaps, to the studies of Bunting in this field. Bunting has emphasized as the characteristic, almost pathognomonic, leukocytic feature a relative, and, as a rule, also an absolute increase in the large mononuclear and transitional cells. This finding would appear to be present throughout the course of the disease, though special emphasis has been laid upon the mononuclear increase in the early stages. The other characteristic features, according to Bunting, are the increase in the number of blood-platelets, many of which are abnormally large, and the presence of detached pseudopodia of megalokaryocytes.

We believe that in many cases of Hodgkin's disease, especially at the onset, there occurs a relative, and often absolute, increase in the large mononuclear and transitional cells, and incidentally, also, of the lymphocytes, but that as the process advances and especially in its active phases this mononuclear increase does not obtain. The particular case we are considering, which, as the study of the histopathology will show, is an advanced one, is illustrative of this. Conversely, however, we do not subscribe to the view that an increase in the large mononuclear cells, even in the early stages of a process characterized by enlargement of the lymphatic nodes, is necessarily diagnostic of Hodgkin's disease. A similar increase is observed not infrequently in tuberculosis and in metastatic carcinoma of the lymph-nodes. We have recently seen a striking example of this in the case of a patient who before the completion of our examination strongly suggested the possibility of a Hodgkin's disease. The leukocytes were about normal in number and the large mononuclears and transitionals varied between 10 and 17 per cent. An excised gland showed a characteristic tuberculous granulation tissue.

The case under consideration stands forth particularly, from the hematologic point of view, by reason of the enormous leukocytosis. The total white corpuscle counts in 70 collected cases serve to emphasize this feature: 22 of these 70 cases showed a leukocyte count of 5500 to 10,000; 20, 10,000 to 20,000; 8, 20,000 to 30,000; 2, over 30,000; and 18, a leukopenia of 5500 to 2000. The data of some observers would indicate, however, that counts above 30,000 are relatively more common than in the preceding table.

The high total in our case is exceptional therefore, and is to be explained, perhaps, by the fact that the disease was very active during the greater part of the time it was under observation, and also by the secondary rôle probably played by the streptococcus, a factor to which we shall return later. (See Autopsy.)

This case is unusual, further, in the constantly low eosinophil count. The eosinophils are variable in number in different cases, but the rule is perhaps a moderate increase in these cells. It is frequently the case, furthermore, that the relative number of polynuclear neutrophils, lymphocytes, and eosinophils runs parallel, more or less, to the number of the same cells in the tissue. Here again the rule has been violated, as we observe numerous eosinophils in the tissue of the excised glands. To explain the almost constant absence of eosinophils in our case, we must take into account the fact that the high total white cell count and the terminal streptococcus infection indicate that the Hodgkin process was overshadowed for a considerable period by a secondary pyogenic infection; and it is well known that the majority of acute infections, in their active phases, are characterized by the absence of the eosinophils from the circulating blood.

In brief, we must repeat, as regards the hematologic picture, what we have noted with respect to other features of Hodgkin's disease—viz., it is highly variable. Many writers state there are no blood changes or that such changes as may occur are not characteristic. So far as formulating a rule goes, we may say that the leukocytic blood-picture is likely to be a practically

normal count—or possibly a moderate leukopenia or a slight leukocytosis—in the early stages of the disease, associated with a relative lymphocytosis, and a variably high leukocytosis of the polynuclear type, with a marked and progressive diminution of the lymphocytes in the advanced stages; while, in addition, an eosinophilia is apt to be present. The leukocyte count is likely, moreover, to be independent of the type of the disease present, whether local or general, febrile or non-febrile, depending upon whether the specific gland process is the controlling factor, or whether the assumed secondary pyogenic infection is of moderate severity, leading to a leukocytosis, or overwhelming, giving rise to a leukopenia.

Hodgkin's Disease and Tuberculosis.—The question as to whether the tubercle bacillus is the specific cause of Hodgkin's disease cannot be said even yet to have been definitely settled, though the consensus of opinion surely leans from that view. We may say to those who uphold the specific rôle of the tubercle bacillus:

1. The finding of the bacillus in Hodgkin's tissue is certainly exceptional.

2. The production, in experimental animals, of tubercle by the injection of emulsions prepared from excised glands has had little confirmation.

3. The association in the same lymph-node of Hodgkin's and tuberculous granulation tissue must be of very rare occurrence.

4. Tuberculous lesions elsewhere in the bodies of Hodgkin's patients are not more frequent than in those who have died from other causes.

5. The possibility that the bacillus of bovine tuberculosis is the specific factor needs further confirmation.

6. The so-called granular form of the tubercle bacillus found in glands of Hodgkin's disease is not the tubercle bacillus, but is identical with the diphtheroid bacillus on which the attention of bacteriologists and clinicians has been focused in recent years. The diphtheroid bacillus, moreover, is no longer looked upon as being the specific factor in the disease.

7. To assume that Hodgkin's disease is due to an attenuated tubercle bacillus or to the toxin of the tubercle bacillus is only to beg the question.

The records in the case before us show the following data relative to a possible tuberculous etiology: 1. The history and environment of the patient were negative as to tuberculosis. 2. The physical examination of the chest (until the terminal, bilateral pleuritis) and repeated examinations of the sputa were negative. 3. There was no reaction to hypodermic injections of Koch's O. T. in increasing doses up to 5 milligrams (the tests were made during an afebrile period). 4. Macroscopically and microscopically the tissues of excised lymph-nodes and of the spleen and other organs removed at the autopsy revealed no tuberculous process. 5. At section there was found only a tuberculous scar of the right apex. 6. Injections of lymph-node emulsions into guinea-pigs did not produce tuberculosis. The use of rabbits as experimental animals, in view of their greater susceptibility to bovine than to human tuberculosis, was not made; nor was the subcutaneous tuberculin test carried out with material made from the bacillus of bovine tuberculosis. However, assembling all our data, including those of the postmortem, we feel that we are justified in concluding that there exists no evidence in this case, at least, of a specific tuberculous etiology.

We may add, incidentally, that the Wassermann reaction in the blood was negative.

Histopathology.—The morbid process in Hodgkin's disease may conveniently be subdivided into three stages, though the interweaving of these periods may be so marked as to make such a subdivision purely a matter of convenience.

In the first stage the microscopic picture tends to be dominated by a hyperplasia of lymphoid cells arising from the germinal centers of the lymph-nodes. Even at this early period, however, there may be seen in variable number those other types of cells—endothelial cells with large nuclei or multinuclear, eosinophils, polymorphonuclears, plasma cells, etc.—which go to make up the characteristic granulation tissue.

The second or intermediate stage exhibits, in a way, the most

exquisite picture of the Hodgkin process. As a transitional phase from stage one we observe a regression of the lymphocytic infiltration; and as a phase leading to the third stage numerous fibroblasts and even young connective tissue. The active granulation tissue proper is made up of endothelioid cells of variable size, some with single large vesicle-like nuclei, others

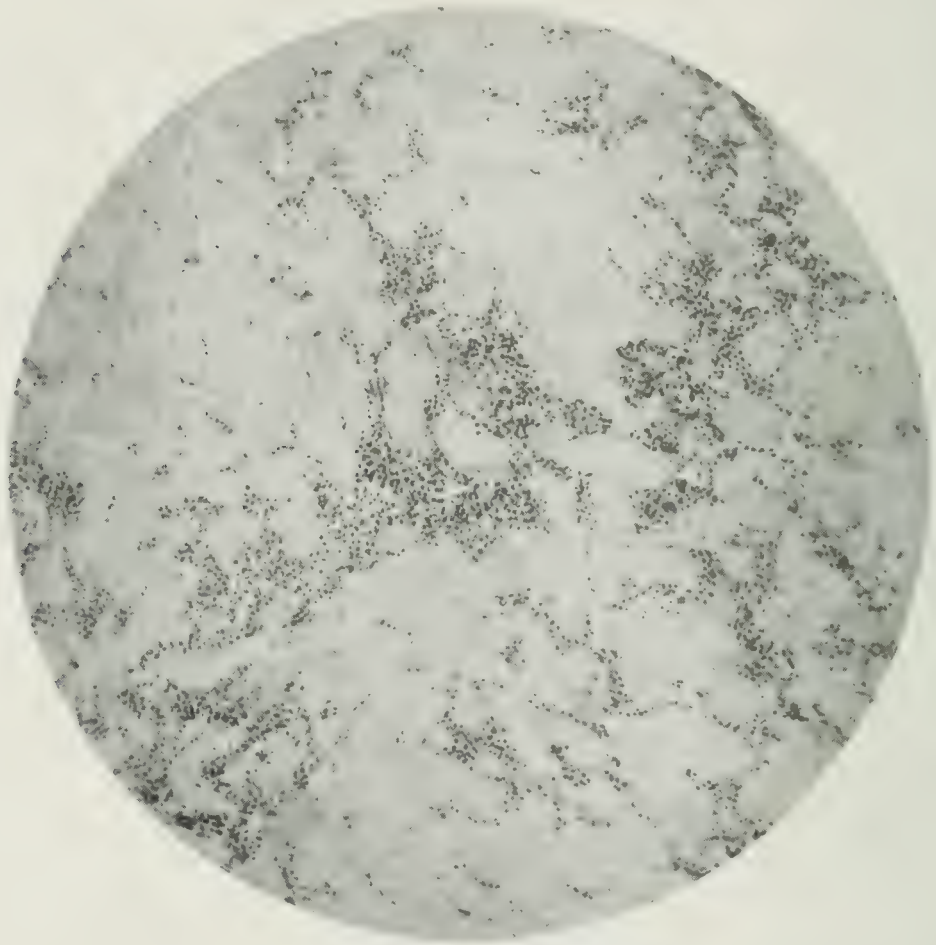


Fig. 41.—A section illustrative of well-advanced changes. To a great extent hyaline connective tissue has replaced the cellular elements. Among the latter a few of the giant type are still to be made out. $\times 115$.

with polymorphous nuclei, and still others with multiple nuclei. These are the giant cells, which form perhaps the most striking feature of the histologic specimen. Many of these cells are undergoing mitosis. Oftentimes these endothelioid cells are so large and so closely packed as to form rods or strands of cells traversing the slide.

The richness and multiplicity of the cell picture is enhanced further by the presence, in the majority of cases, of many eosinophils, considered by some observers as the most diagnostic of the cellular type. Scattered among the endothelioid cells of bizarre forms and among eosinophils—the whole with no definite arrangement—are polynuclear neutrophils, lymphocytes,



Fig. 42.—A section showing phases in the fibrous metamorphosis not as advanced as in Fig. 41. Above is seen a multinucleated giant cell. $\times 230$.

plasma cells, and cells of the proliferating reticulum. The picture once seen and studied cannot be forgotten.

The third and last stage of the process is itself subject to subdivision into the phase in which necrosis, with the formation of a cellular débris, takes place, and the phase in which the cellular elements are more or less completely replaced by a hyaline connective tissue.

The sections from our case show only the transition between stages two and three and well-marked evidence at many places of stage three (Figs. 41-44).

Types and Course of the Disease.—*Termination.*—This case has pursued what may be called the usual course, except, possibly, in the matter of rapidity. From the time when the first

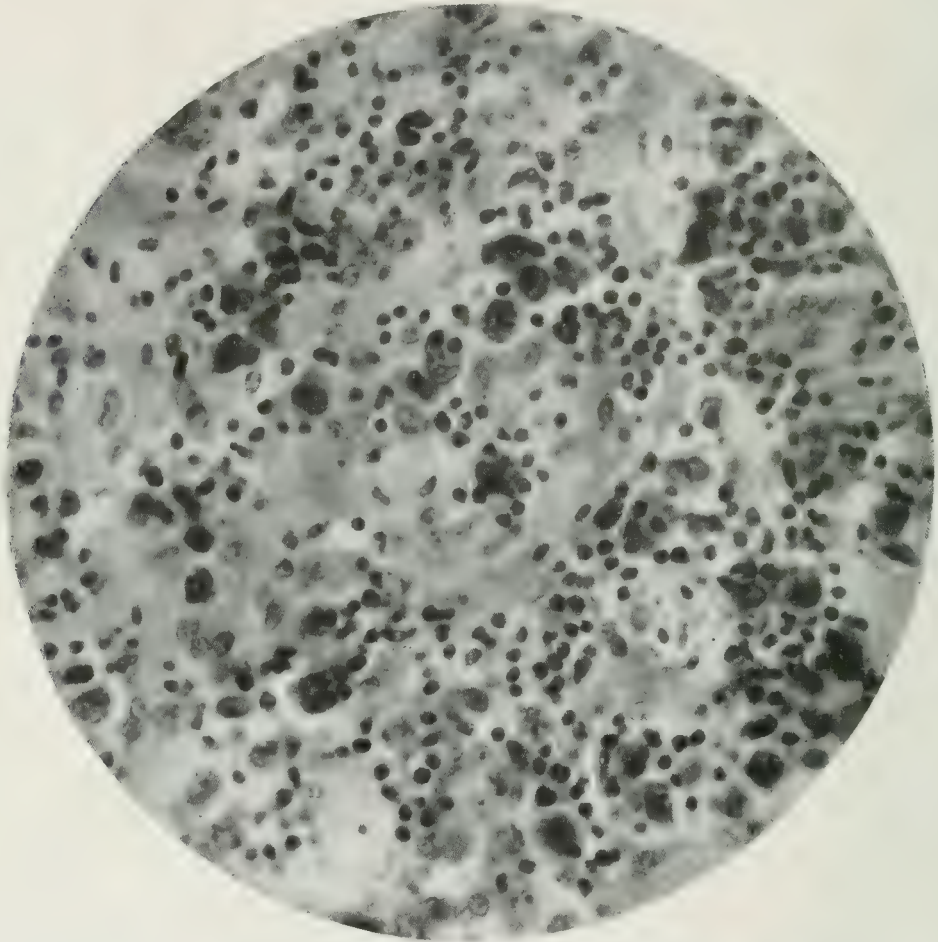


Fig. 43.—The various cell types characteristic of the disease are present in this section, though the eosinophilic granules of some are not reproduced. $\times 325$.

cervical node was noticed until exitus was a matter of approximately a year. The course was usual in that lymphatic enlargement began in one cervical region, gradually involved the remainder of the nodes on the same side, and then on the other, and finally became general. Other less frequently observed types are: 1. The *acute*, in which there is a rapid involvement of most

of the lymphatic nodes, subsequent to the beginning in one group or another, and a rapidly fatal termination. 2. The *indolent, localized form*, in which the process is confined to a single group for a long period and becomes more general late in the course. 3. The type in which the lymphatic involvement is primarily of the deep nodes, particularly the retroperitoneal—



Fig. 44.—A higher magnification of a portion of Fig. 43. $\times 400$.

a form characterized by febrile phenomena and progressive cachexia and often difficult of recognition until the appearance of changes in the superficial glands (*typhoidal form*). 4. The *splenic type*, in which changes in the spleen predominate. 5. Possibly *mycosis fungoides* and *Mikulicz's disease*.

About a month before death the clinical picture in this case underwent a rather striking change. The patient began to complain of pain in both sides of the chest, dyspnea became marked,

the presence of fluid in the abdomen could be determined, the temperature sought a higher level, and the man's appearance suggested sepsis. Paracentesis of the chest yielded, on both sides, a fluid with the usual characteristics of an exudate, the cells of which were 65 per cent. polymorphonuclear. A smear from the same showed the *Streptococcus hemolyticus*. A node removed at this period, upon culture, showed a hemolytic streptococcus, as did two blood-cultures. In a word, a streptococcus septicemia dominated the scene in its later stages.

The Autopsy.—The postmortem examination added what verification was needed of the nature of the process present and of its complications. The only evidence of tuberculosis was the healed lesion (scar) of the right apex. All of the lymphatic nodes were enlarged, the mediastinal most, the mesenteric least. The glands were firm, even hard, and on section were slate-gray in color, showing considerable calcification in places, but nowhere caseation. A mediastinal packet was adherent to the sternum, pericardium and left pleura, and extended into the left lung, causing a pressure atelectasis of the upper lobe.

The spleen weighed 530 grams and was studded with walnut-size tumors, on section resembling the appearance of lymph-nodes. The consistency of the organ was decreased (acute septic splenitis), its color on section grayish white, the pulp prominent, and the trabeculae invisible. A fibrinous perisplenitis was also present.

The pleural and peritoneal cavities contained a serofibrinous fluid; and the liver was covered with the same fibrinous material as the spleen.

The *Streptococcus hemolyticus* was cultivated—and was also present in smears—from the heart blood, lymph-nodes, spleen, and the fibrinous exudate on the pleural and peritoneal membranes.

The histopathology was that of Hodgkin's disease in all the organs affected. There was no associated tuberculosis in the lymph-nodes, spleen, etc.

Summary.—In résumé, we would emphasize the following aspects of this case of Hodgkin's disease:

1. *Nomenclature*.—Hodgkin's disease is the best available name for this process.

2. The *onset* with pruritus and the appearance, later, of pruriginous lesions.

3. The *hematologic picture*: a total leukocyte count, almost constantly over 30,000, of the polynuclear type, with a relative diminution of the lymphocytes, no increase in the large mononuclear and transitional cells, and an almost complete absence of the eosinophils.

4. The absence of all data pointing to a tuberculous rôle in this case.

5. A typical histopathologic picture, with fibrosis well advanced.

6. A termination indicating apparently a secondary streptococcus infection.

CLINIC OF DR. RICHARD J. TIVNEN

MERCY HOSPITAL

EYE FINDINGS AS AN AID TO THE DIAGNOSIS OF GENERAL CONDITIONS; A SUGGESTION FOR TEAMWORK

WE will consider this morning the value of eye examinations as an aid in general diagnosis. Let us ask ourselves, as ophthalmologists, the frank question, Are we able by our examinations of the eye to obtain information which will be of real practical value to the physician in reaching a diagnosis, suggesting a treatment, and estimating a prognosis? We can answer this question without hesitation in a positive and emphatic affirmative. We can go farther, and say that our contribution in the solving of many perplexing pathologic problems is not alone an important one, but is very often a determining one. It is not unusual for the ophthalmologist to discover in the eye evidences of general disease which had been previously unsuspected. It is likewise a common experience for the ophthalmologist, from the data an eye examination supplies, to be able to estimate a prognosis, in a general condition, with a certainty comparing favorably with any of the modern methods employed for this purpose.

The diagnosis of a diseased condition is the outstanding feature in every case.

In a large percentage of cases, however, the time when—that is the period of development—the condition is recognized is a vital factor in the successful issue of the process. An early diagnosis of a given condition may mean saving the patient's life; a late diagnosis, disaster. The problem, then, is not alone one of diagnosis, but the more important and determining one

of *early diagnosis*. It is precisely in cases in which our early diagnosis may avert distressing if not indeed fatal consequences that often present the greatest and most hopeful opportunity for the exercise of "team-work."

The aid of the ophthalmologists in these later years has been sought far more commonly than formerly. It must be said, however, that there still remains much need for establishing more frequent co-operation, more "team-work" between the ophthalmologist and the physician. This "team-work" cannot fail to be reciprocal—the physician obtaining much valuable and practical information from the ophthalmologist, even from a negative report, while the ophthalmologist may obtain equally valuable information from the physician, even though it be also of a negative character. There are, indeed, many, very many ocular conditions little understood at present which undoubtedly owe their origin and development to obscure constitutional dyscrasias, which must, unless the interest and co-operation of general medical men be enlisted, continue to remain dark chapters in ophthalmology.

The all too prevalent custom of enlisting this co-operation, this "team-work," only after a considerable period of observation has transpired and all other methods of investigation and therapeutic measures have been exhausted, is unwise, and deprives the patient in many instances of the benefit of an early diagnosis, upon which his vision, perhaps his life, may depend. The ultimate goal, therefore, of our efforts in establishing and utilizing this reciprocal relation between the physician and the ophthalmologist should be to create a "routine mental attitude," so to speak, in both, which will serve to suggest at the *beginning* of the investigation of an obscure medical problem the wisdom of enlisting this co-operation in the hope that something of value may be elicited in solving and combating the difficulty. If such early co-operation, such "team-work," were adopted as a routine method of procedure, the results attained could not fail to impress and win the commendation of the patient, the physician, and the specialist.

Let us this morning refer to a number of general conditions

which commonly present ocular manifestations, and which serve to emphasize the value of "team-work" between the physician and the ophthalmologist. We will omit all reference to clinical cases in our consideration in order that a more rapid and general survey of the subject may be presented, and refer in a cursory way only to those diseases discussed, without attempting to establish more than the relation and value of eye examination in such conditions.

The lesions of the brain and meninges present ocular manifestations at various periods in their clinical course which are of special and dependable value to the internist, the neurologist, and the surgeon.

These manifestations are so constant and reliable, it may be stated as an axiom that the investigation of a brain involvement cannot be said to be complete unless an eye examination has been made. A brief reference to the structure of the eye and its relation to the brain, meninges, and cranial cavity is of value in appreciating these manifestations. The globe, optic nerve, and retina, as we know, may be regarded as an extension or outgrowth of the brain. The retina is, in reality, a part of the brain, and the optic nerve is essentially a cerebral tract. The globe is embedded in the orbit and is in intimate relation with the nose, nasal accessory sinuses, and cranial cavity.

Each optic nerve trunk is made up of nerve-fibers separated by neuroglia, collected into bundles and held together by connective tissue; lymph centers occur between the connective tissue and the bundles. The nerve trunk is surrounded by three enveloping sheaths—the dura, arachnoid, and pia—which are the direct continuation of the cerebral meninges. Between the dura and pia is the intervaginal space, which is divided by the arachnoid sheath into two lymph spaces, the subdural and sub-arachnoid. Both of these lymph spaces originate in and are continuous with the corresponding cerebral channels. These enveloping sheaths terminate in the sclerotic coat of the eye. The central vessels (artery and vein) of the retina run in the axis of the nerve to the optic papilla; there they divide and become the retinal vessels. The connection of the cerebral lymph

spaces with the sheaths enveloping the optic nerves explains the readiness with which an increase of intracranial pressure may affect the nerve and retinal vessels and produce ocular manifestations. The two optic nerve trunks proceed backward from the globe and join at the *chiasm*. At the chiasm the fibers from each nerve trunk semidecussate, forming a right and left *optic tract*, which diverge and proceed backward respectively to the right and left primary subcortical optic centers and visual areas in the occipital lobe. The nerve-fibers of accommodation—convergence and pupillary contraction—pass from the optic tract to the oculomotor nucleus, and from this nucleus send fibers in the trunk of the oculomotor nerve to the pupil (sphincter iridis), to the muscle of accommodation (ciliary muscle), and to the converging muscle (internal rectus). This semidecussation of nerve-fibers and distribution of tracts explains the various pupillary, accommodation, convergence, and hemianopic phenomena frequently exhibited in cerebral neoplasms and other intracranial disturbances which often provides important data in diagnosis and localization of the lesion. The chiasm rests upon the sphenoid bone and is in intimate relation with the pituitary body, the anterior end of the third ventricle, the infundibulum, internal carotids, and the meninges. In addition to the foregoing anatomic relations, it must be recalled that the eye is an important and exceedingly sensitive part of the central nervous system. Its special function—vision—is a highly specialized act; and the optical and neuromuscular elements engaged in this delicate function exercise a wide and exacting influence on the general economy, and is itself highly susceptible to even slight disturbances of a local or systemic origin.

With this brief and superficial reference to the structure and relations of the eye, we will now consider a few conditions presenting ocular manifestations commonly met with by the ophthalmologists which will serve to emphasize the value of co-operation and "team-work."

Tumors of the brain present, as a rule, some time in their development ocular manifestations which may be of great and determining value in aiding the diagnosis, gauging the clinical

progress, estimating the prognosis, determining the localization, and suggesting the treatment. "Choked disk" is encountered in 80 to 90 per cent. of such cases, and it is accepted as the most common general symptom of brain tumor and one of the most constant symptoms of tumor of the cerebellum. The precise cause of the choked disk remains as yet to be determined, though the mechanical theory is most generally accepted. This theory assumes that the intracranial pressure is raised as the growth enlarges and the cerebrospinal fluid is pushed from the lymph spaces of the meninges into the corresponding spaces between the sheaths surrounding the optic nerve causing distention of the sheath, edema of the nerve head, and venous stasis. The presence of a choked disk indicates increased intracranial pressure, and in the majority of cases, other causes being excluded, a brain tumor. It is of importance to emphasize that the external appearance of the eyes and the patient's central vision may be practically normal even though the nerve head exhibits a well-marked choked disk. Hence, it is well to remember that the patient's vision or the external appearances of the eyes are no indication of the presence or absence of this symptom. The changes in the disk, disclosed by repeated examinations, supplies dependable data upon which one may reckon the clinical progress of the case and base the decision of a decompression operation.

In a suspected case of brain tumor the absence of a choked disk—unless other symptoms point clearly to the presence of a brain neoplasm—causes a grave doubt as to the correctness of the suspicion.

Too much emphasis cannot be given to the suggestion that early and repeated eye examinations should be made in this class of cases. It is a common experience for the ophthalmologist to see such patients with a well-marked choked disk or, indeed, presenting a secondary optic atrophy consequent upon continued and unrelieved increased intracranial pressure. Such patients are doomed to blindness or greatly impaired vision. It is certain that in many of them useful vision might have been preserved had the ocular condition been recognized earlier and a proper operative procedure instituted. In addition to the symp-

tom of choked disk, other evidences of brain tumor are frequently manifested in the ocular structures, such as alterations in the form and color fields, pupillary phenomena, ocular palsies, diplopia, nystagmus, etc. It is frequently possible to assist in localizing the brain lesion by means of perimetric examinations for form and color, study of the pupillary and other eye symptoms.

Diseases of the meninges very frequently exhibit characteristic ocular changes affecting the pupils, muscles of the lids and globe, vision, nerve head, retina, and choroid. The ocular manifestations are, as might be anticipated, dependent in degree and period of development upon the virulence of the process and, in particular, upon its location, being slight and late when the meningitis involves the convexity, and extensive and relatively early in the basal types. Many cases of meningitis, particularly in their incipency, present considerable difficulty in diagnosis. It is a common experience that pneumonia and typhoid fever in the early stages simulate a meningitis and render a diagnosis exceedingly difficult.

The eye findings—particularly repeated observations—in such cases may be of great value in differentiating. In the tubercular forms tubercles in the choroid when present is a symptom of considerable diagnostic and prognostic importance.

In *head injuries* and *skull fractures* ocular manifestations chiefly of the nerve head, pupillary symptoms, provide important information in estimating the presence and degree of intracranial pressure, and also in some instances aid in localizing the site of the injury. In such cases the state and progress of the intracranial pressure, estimated by repeated ocular observations, may be of importance in deciding whether an operative procedure be instituted to avert compression of the medulla.

The *eye symptoms of tabes* are well recognized and among the most important and dependable symptoms of this disease.

Very frequently they are the only symptoms presented which indicate clearly the nature of the difficulty and establish the diagnosis. Argyll Roberston pupil is one of the most constant symptoms, occurring, according to some authors, in 76 per

cent. of cases. The value of the symptom is enhanced by the fact that it is often a very early manifestation.

Optic atrophy is often an early symptom and is found in approximately 20 per cent. of cases.

Ocular palsies and alterations in the visual fields, particularly those for color, are usually present and are of considerable diagnostic value.

Disorders of the pituitary body commonly present ocular symptoms which are often first discovered by the ophthalmologist, and disclose the nature of the malady. The symptoms presented are chiefly those affecting the visual fields, exhibiting varieties of hemianopsias; amblyopia, exophthalmos, pupillary phenomena, and nystagmus are also encountered, depending upon the character, location, and progress of the lesion.

In *nephritis* many ocular changes occur which are of considerable value as an aid to diagnosis and prognosis. It is a common experience for this condition to be first disclosed by an ocular examination.

The usual and most important ocular manifestation is that of the so-called albuminuric retinitis, which occurs, according to some authors, in 20 per cent. of cases. It occurs most frequently in the small contracted kidney, next in the chronic diffuse parenchymatous forms, next in the nephritis of scarlatina, and least in cases of amyloid degeneration. From a prognostic standpoint the presence of a well-marked albuminuric retinitis in cases of chronic nephritis is of grave import. It is commonly stated that 85 per cent. of such cases die within two years. Considerable reservation, however, in accepting this dictum is necessary, as it has been found that in many cases who have pursued a strict régime and are equipped with sufficient means to insure their comforts, are able to prolong life for considerable periods beyond these limits.

No class of cases, perhaps, emphasizes the value and necessity of co-operation between internist and ophthalmologist more strikingly than cases of Bright's disease.

Diabetes.—The ocular manifestations of diabetes are many

and varied, involving the lens, retina, external ocular muscles, cornea, iris, ciliary body, vitreous, choroid, and optic nerve.

Cataract is said to occur in from 4 to 25 per cent. of cases.

Myopia appearing in patients past forty is often of the so-called diabetic-myopia type. Urinalysis is always indicated in such cases.

Diabetic retinitis is usually a late development of the disease, and when accompanied by extensive retinal hemorrhages indicates a grave prognosis. It is not an unusual experience for patients complaining of "failing vision" to present themselves for relief to the ophthalmologist, who discovers that the cause of their visual difficulties is an unsuspected diabetes or nephritis.

Syphilis.—All the structures of the eye, with the exception of the lens, may be affected with syphilis in any of its stages. Alexander reports that 2.16 per cent. of eye diseases are due to syphilis. The uveal tract is the most common seat of the infection. Slight injuries of the eye in syphilitic subjects frequently develop pathologic ocular processes out of all proportion normally expected from slight traumas.

Tuberculosis.—Primary tuberculosis is of infrequent occurrence as an ocular affection. The ocular lesions are almost always of the secondary type. Lupus is met with upon the lid.

During the course of a general tuberculosis the ocular structures may be attacked, most commonly the uveal tract. Phlyctenular conjunctivitis, so common in children of the so-called "scrofulous diathesis," is held by many observers to be of tubercular origin.

Vascular Retinal Changes.—An ophthalmoscopic examination often reveals distinct alterations in the retinal vessels consequent upon a vasculitis and perivasculitis. Such changes often give the first intimation of a systemic disorder, such as a chronic nephritis or general arteriosclerosis. Their presence is of grave import, and may be the early manifestations of an impending cerebral sclerosis or hemorrhage.

Marcus Gunn states that "ophthalmoscopic examination is one of the most ready clinical means for the early detection of important arterial changes."

deSchweinitz considers the triad of a corkscrew-like appearance of the smaller retinal arterial vessels, the flattening of a retinal vein where it is crossed by an artery, and a general congestion of the optic disk as almost pathognomonic of the early stages of arteriosclerosis, and he places great value upon the ophthalmoscopic examination as an aid in the determination of persistent high arterial tension.

Eye-strain.—No group of distressing and baffling disorders offers a larger field for real co-operation, "team-work," than patients suffering from eye-strain. It might also be added that there is no class of cases in whom more distinctly beneficial results is attained by such co-operation. Very frequently sufferers from low degree of refractive errors complain of symptoms which completely disguise the origin of their difficulty as being due to eye-strain.

The literature and one's experience abounds with most extraordinary reports of such cases whose difficulties had been diagnosed as migraine, vertigo, neuralgia, cerebral anemia, cerebral congestion, brain tumor, nervous breakdown, appendicitis, and other maladies. Many of the patients afflicted with eye-strain have been labeled neurasthenics, neurotics, hypochondriacs, and, it must be admitted, many of these, being denied serious attention from the medical profession, have sought relief in the sanctuaries of the Christian Scientist and other cults.

A characteristic feature of this class of cases is that seldom do they associate their headaches and other symptoms with their eyes, and few of them have impaired vision.

Their error of refraction usually is of the low grade, astigmatic type, and the correcting lens required is of very low degree. This class of patients are the *bête noir* of the internist, the neurologist, the gynecologist, and the surgeon.

Their complaints are grotesque and inconsistent, their manner and behavior depressing and pathetic, and their symptom-complex almost incomprehensible.

The prompt and satisfactory relief many of these cases obtain from properly fitted glasses is remarkable, and suggest

unmistakably the wisdom of co-operation with the ophthalmologist in their management.

The foregoing brief and general review of our subject is merely a suggestion of the great value of co-operation and "team-work."

While we as ophthalmologists already have very definite information of various ocular manifestations of general diseases which undoubtedly could be utilized by our confrères, it is certain that a superlative degree of benefit would result to our specialty if a more frequent, intimate, and routine co-operation and interchange of opinion, investigation, and observation existed.

CLINIC OF DR. ROBERT SONNENSCHN

COOK COUNTY HOSPITAL

SOME INTERESTING EAR CASES

FROM the material at hand we will be able this afternoon to show you a number of ear cases presenting some interesting features. These, I trust, will be of value to you not only from the standpoint of diagnosis, but from the practical side of determining when certain lines of treatment are to be instituted and when not.

Our first case is a surgical one, in that a radical mastoid operation was performed.

Mr. G. N. M., aged nineteen, student, gave a history of having had an intermittent discharge from both ears, especially the right, during the past five or six years, more marked during the winter. At the time he first presented himself he claimed there had been for two weeks a bloody, purulent discharge from the right ear, accompanied by dull headache radiating from the ear to the occipital region and into the shoulder. Hearing had been considerably impaired and a buzzing tinnitus was present. Tonsils and adenoids were removed two years ago.

Examination made when the patient was first seen two months ago: The nose showed considerable mucopus in the right nares; pharynx had some swollen, very red, lymphoid tissue on the posterior wall.

The ears: Left drum membrane was markedly retracted and atrophic. The right drum-head was scarred and adherent to the inner tympanic wall; there was a marginal perforation in the posterior superior quadrant, but no pus discernible. Hearing for whisper $1\frac{1}{2}$ meters on each side. The tuning-fork tests show that the Weber was lateralized on the right side, and there was diminished hearing for all the octaves, with failure on the right side to hear the C-1 (32 double vibrations).

The diagnosis made at that time was acute exacerbation of a chronic otitis media. The usual treatment of rest, phenol-glycerin pack, and 50 per cent. alcohol dressing was instituted; but no improvement in the pain noted. The ear remained dry throughout the entire period. In view of the fact that intracranial complications, such as extradural abscess, meningitis, brain abscess, etc., in the course of a chronic otitis media, usually occur at the time of an acute exacerbation, the patient was placed in the hospital and carefully observed for some weeks. The eyes were repeatedly examined, but no choked disk or other fundus changes found. The Wassermann reaction was negative. Several neurologic examinations were made, but each time the report was that "no intracranial findings could be noted." The temperature was usually normal; at times subnormal, and only occasionally rose as high as 99° or 99.2° F.

The slow pulse shown by the patient was most noticeable. It ranged practically all the time from 48 to 68 per minute. The patient stated that during an attack of influenza which he experienced in the fall of 1918 his pulse was found to be very slow. The heart showed a systolic murmur loudest over the aorta. The leukocyte count was made every second day for several weeks, and ranged from 10,000 to 15,000; the polymorphonuclear neutrophils showing from 66 to 76 per cent.

x-Ray pictures of the nasal accessory sinuses showed some cloudiness of the left maxillary antrum, but irrigation of both antra revealed no pus. The radiograph of the head showed a marked density of the right mastoid region.

The Labyrinth Tests.—Rotating patient to the right ten times gave after-nystagmus to the left lasting thirty-five seconds; turning to the left gave nystagmus to the right lasting twenty-seven seconds. Owing to the perforation in the drum membrane it was not deemed advisable to try the caloric test. The past-pointing to the left was somewhat abnormal, but to the right it was normal.

The pain and disturbance of sleep continued despite complete rest in bed, careful diet, free catharsis, alcohol dressings to the mastoid region, etc.; the tenderness to pressure remained fairly

constant in the lower parietal and in the mastoid region on the right side.

The continuous pain, tenderness, slow pulse, and leukocytosis pointed to a suppurative process, and seemed to indicate the presence of intra- or extradural irritation. In view of these signs and symptoms, and the fact that the patient was steadily getting worse, a radical mastoid operation was performed. On chiseling through the cortex the lateral sinus was found lying directly over the antrum, so that despite all possible care it was opened and then had to be packed. The dura of the middle fossa was exposed, but found normal. The tip of the mastoid was found filled with granulations; these were removed, and after the typical radical mastoid had been completed a Panse plastic was made. A very small silkworm-gut drain was inserted in the line of incision and removed a few days later.

There was almost immediate relief from the headache and an otherwise uneventful recovery. The patient's appearance has rapidly improved and he will now leave the hospital feeling fine. The slow pulse is still present and we can account for it only on the ground that it is a personal peculiarity of this individual. We know that in some families a slow pulse is present in many of the members. The very interesting and instructive fact in this case is the presence of symptoms pointing to a marked dural irritation during the course of the acute exacerbation of a chronic otitis media, and that at the time of operation the only pathologic condition found was that in the tip of the mastoid; while we could at the time not explain all the symptoms on the basis of this change alone, we do know that cleaning out the mastoid relieved the patient of the long-standing symptoms he had had.

We will now examine a patient presenting symptoms referable almost entirely to the vestibular apparatus rather than to the hearing portion of the ear.

CASE II.—Mrs. E. C. W., aged twenty-eight years, a housewife. The patient had one child several years ago and is now four months pregnant. She complains of occipital headaches

which occur especially during the day; examination of the eyes made ten months ago, so she says, showed some muscular disturbances, but no other findings. During the past month there has been some cough and at times difficulty in breathing. The main reason for the patient presenting herself today is that during the past five years, and especially during the last year, there has been a tendency at times to fall forward and to the right; nausea, vomiting, and vertigo are also present during these attacks. Several months may elapse without any of these symptoms recurring. Sudden movements of the head or body cause a tendency to fall, and give the patient the sensation as though surrounding objects were moving about in a circle. There is no impairment of hearing, but the patient at times notices a low-pitched tinnitus aurium.

Examination.—The nose shows a septum markedly deviated to the right. The pharynx and tonsils are submerged, with some pus in the right one.

The Reflexes.—Corneal and pharyngeal are much diminished, while the patellar is considerably increased.

The Ears.—Drum membranes are negative. The hearing for the whispered voice is 8 meters on both sides. All the tuning-forks, from the lowest to the highest octaves, as well as the high tones of the Galton whistle, are well heard. The Rinné is positive (*i. e.*, the air conduction is longer than bone conduction) and the Weber is not lateralized. The only abnormality is a slight diminution in the bone conduction as shown by the Schwabach test. The caloric reaction is most striking, in that less than 120 c.c. of cold water instilled into the ears produces very speedily a rotary nystagmus to the opposite side with intense vertigo and nausea. The past-pointing is normal with both hands. There is no inco-ordination, as demonstrated by the Romberg and pointing tests, and no spontaneous nystagmus.

We thus have here a perfectly normal cochlear function as shown by the hearing tests, and a hypersensitive vestibular apparatus, as seen in the very decided reaction to the small amount of cold water. Owing to the patient's highly excited condition no further tests, such as the turning, etc., can be made.

The change in the corneal and other reflexes would point somewhat to a functional disturbance, possibly a neurasthenia or hysteria. In the former we often have a vestibular apparatus which responds very markedly to all stimuli.

It is very important in all cases where vertigo is complained of by the patients not to suggest in our questions anything leading, but to allow the individual to describe the symptoms himself. Often the vertigo is described as a "feeling of faintness," or that "things appear black," or that "spots dance before the eyes," or that there "is a sensation of sinking." These are due, as a rule, to syncope, gastro-intestinal, or other disturbances. In the real labyrinthine vertigo the person either feels like falling or actually does fall in one direction or the other; namely, in the direction of the slow component of the nystagmus, while the surrounding objects appear to move in the opposite direction; namely, that of the rapid component of the nystagmus.

In this otherwise healthy person there is apparently some undue irritability of the vestibular apparatus, the source of which is not clearly demonstrable. We shall suggest the use of bromids in the effort to quiet the nervous system; and allow some time to elapse to see the effects of this medication. Ordinarily we would use large doses, but in view of the pregnancy we will request the obstetrician to determine the amount which can safely be taken.

Our next patient will illustrate a serious involvement of the sound-conducting mechanism of the ear.

CASE III.—Mrs. S. J., aged thirty-one, a housewife. This patient comes here complaining of markedly impaired hearing and high-pitched tinnitus, especially in the right ear, for the past four years. There has been no discharge from the ears at any time, and there is no history of impaired hearing in the family. The hearing became worse during the last pregnancy. There is no nasal obstruction, but some time ago adenoids were removed for the purpose of improving the hearing, but without producing any such results. Repeated ear treatments, the patient states, have also failed to help her.

Examination.—The nose shows a septal deviation to the left and also polypoidal middle turbinates. The nasopharynx shows some very small adenoid remnants. The tonsils are submerged and there is some purulent secretion in the crypts.

The Ears.—The drum membranes are somewhat dull and slightly opaque. Hearing tests: The whispered voice is heard at a distance of only 20 cm. With tuning-forks we find that the three lowest octaves are not heard in either ear; the highest fork c^5 (4096 double vibrations) is heard with diminished intensity on both sides, especially the right. The Weber is not lateralized, the Rinne is negative (that is, bone conduction is longer than air conduction), and the Schwabach is increased (*i. e.*, bone conduction is longer than in the normal individual). The eustachian tubes are open, but catheterization does not improve the hearing.

The dulness and opacity of the drum membrane might lead one to suppose that a tubal or middle-ear catarrh had been present, but the patency of the eustachian tubes on catheterization disproves this. The presence of Bezold's triad—loss of hearing for the low tones, a negative Rinne, and a lengthened bone conduction—stamps the case as one of otosclerosis. The characteristic of this disease is also the fact that it occurs mostly in young women and is made worse by each pregnancy, as shown in this case. The term "otosclerosis" is a misnomer and a very misleading one. There is really no sclerosis or hardening in the ear, as the name would suggest, and as so many men believe. What actually occurs is a spongification of the labyrinth capsule—an absorption of the bone and its replacement by new vascular bone. In the typical otosclerosis this process occurs in the region of the oval window, with a resulting partial or complete ankylosis of the foot plate of the stapes; we then have the symptoms of a conduction deafness giving Bezold's triad, as mentioned above. In the atypical variety this spongification takes place in other parts of the bony labyrinth wall, producing degeneration of various portions of the organ of Corti, with symptoms of a nerve or perception deafness.

In either form of this disease a knowledge of these pathologic

changes will at once show the futility of operative procedures either in the nasopharynx as the removal of adenoids in this case, or tonsillectomy as done by some men, or of intranasal operations, as in the next case which we will show. Unfortunately, the correct diagnosis is not made in the first instance. Mere inspection will not suffice, for the drum membranes often appear normal or show at times only a faint, rosy glow in the posterior half. A thorough tuning-fork examination is needed and should be made to accurately establish the diagnosis. All manner of medicinal, mechanical, and electric measures have been used, but instead of aiding they often hasten the progress of the disease.

Phosphorus in doses of $\frac{1}{100}$ grain three times a day over a period of several months has sometimes been of service; the rationale of its employment having been the theory that otosclerosis is similar in its pathology to osteomalacia.

The next patient also has this so-called otosclerosis, together with certain nasal symptoms.

CASE IV.—Miss A. J., aged twenty-one, a seamstress, presents herself with a history of having had greatly impaired hearing for the last ten years. Tinnitus aurium is present at times. A specialist who was consulted some years ago made the diagnosis of "catarrh of the ear," stated that catheterization was indicated, but that inflation of the ears could not be properly performed owing to nasal obstruction due to large inferior turbinates. A nasal operation was advised and carried out. This, however, together with the catheterization, did not give any improvement in hearing. The sense of smell has gradually disappeared, and there is considerable obstruction to breathing due to the accumulation of large crusts.

Examination.—The nose shows that the inferior turbinate bodies have been almost entirely removed, producing very large nasal spaces. The mucosa is pale and atrophic and there is considerable crusting, especially over the stump of the right inferior turbinate, together with a rather foul odor.

The Ears.—The drum membranes are negative. The whispered voice is heard at 1 meter on the right side, but only

15 cm. on the left. The tuning-forks show negative Rinné, lengthened Schwabach (bone conduction), and no lateralization of the Weber. The C-1 (32 double vibrations) was not perceived in either ear, and all the other octaves were heard with diminished intensity as compared with the normal. On catheterization the eustachian tubes are found widely open, but there is no improvement in hearing from the inflation.

Here we have again a case of otosclerosis characterized by negative drum membranes, greatly impaired hearing, especially for low tones, tinnitus aurium, lengthened bone conduction, and negative Rinné. We see here the sad results of the lack of proper diagnosis in the first instance, when the patient was examined several years ago. Had careful tuning-fork tests been made, a tubal catarrh could not have been diagnosed, but even if the latter had been present, there would have been no justification for the removal of so much of the inferior turbinates. The mucosa of the respiratory portion of the nose is very vascular, and is covered with a columnar ciliated epithelium. When this is removed it is replaced by a flat squamous cell, and the function of the turbinates—which is largely that of warming, moistening, and filtering the inspired air—is perverted. Abnormally large nares are created by this excessive turbinectomy, the secretions dry, form crusts which adhere, and often decompose, producing a foul odor or ozena.

If in a tubal catarrh such extensive operations are contraindicated, how much more so when a diagnosis of otosclerosis with fixation of the stapedial foot plate is made, as in this case. As shown in the previous patient, most lines of treatment are of no avail, and particularly operations, be they performed in the nose, nasopharynx, or pharynx. In the patient before us a most distressing intranasal condition was added to an already very unfortunate irremediable ear impairment because of a lack of accurate diagnosis.

The last case which we will bring before you today presents very serious impairment of both the hearing and vestibular functions of the ear, possibly due to tumor formation in the cerebellopontine angle.

CASE V.—Mrs. M. D., aged thirty-six, a housewife. This patient was first seen a week ago, and at that time stated that at intervals during the past three years she has had attacks of vertigo, nausea, and even vomiting. Surrounding objects move toward the right; the direction of falling tendency is not changed by the difference in the direction of the head. The vertigo is said to be worse during menstruation, and its onset is usually preceded by insomnia for several nights. The present attack of this symptom-complex had been noticed for three weeks, and had been especially severe during the last few days before we first saw her. For some time there has been greatly impaired hearing in the left ear, but without tinnitus or discharge. Tonsillectomy was performed nearly two years ago.

Examination.—The nose shows a small septal crest on the left side.

Reflexes.—Pharyngeal and corneal (on both sides) greatly diminished. Patellar reflex much increased on both sides. Slight horizontal nystagmus on looking to the right.

Ears.—Drum membrane dull; whispered voice heard in right ear at 8 meters; in the left ear not at all. Tuning-forks: In the right ear all the octaves are heard; in the left ear none of them are heard. With the Sonnenschein noise apparatus the left ear is found to be deaf to all sounds. Caloric tests: In the right ear 240 c.c. of cold water gave rotatory nystagmus to the left in less than a minute. In the left ear 720 c.c. of cold water gave no response even after three minutes. There was no use in trying the past-pointing reaction in this patient, for if the labyrinth fails to functionate and no nystagmus occurs with the caloric test there will be no response from the cerebellum, for the impulses would not reach that structure from the vestibular apparatus.

The patient was given mixed bromids one week ago in doses of 10 grains four times a day, and on returning this afternoon states that she is feeling very much better. How long it will be before another attack of vertigo, together with the other symptoms, ensues one cannot say.

This case presents a number of very interesting features.

The increased patellar and the diminished corneal and pharyngeal reflexes would suggest a functional disturbance, such as an hysteria, but the other symptoms are of such a nature as to lead one to the conclusion that an organic disturbance is present.

The left ear is absolutely deaf to the voice, as shown by the noise apparatus inserted in the right ear and shouting into the left one, and is also deaf to all tuning-forks, from the lowest to the highest octaves. Furthermore, the vestibular apparatus of the left ear fails to respond to the Barany caloric reaction. While the turning test—in which the individual is rotated ten times within twenty seconds and the after-nystagmus noted—is of great value, it does not give definite information regarding one labyrinth alone, since both are stimulated at the same time, but with the caloric test only one labyrinth at a time is caused to react. By irrigating the ear with cold water (at about 68° F.) a rotatory nystagmus to the opposite side is induced (*i. e.*, the rapid component of the nystagmus goes to the opposite side); and with the use of warm water the nystagmus is directed toward the side being irrigated. Normally, for instance, if cold water is instilled into the left ear the rapid component of the nystagmus goes toward the patient's right side, and the past-pointing of both hands would be in the direction of the slow component; namely, toward the left. If, however, the vestibular apparatus fails to functionate, impulses will not reach the cerebellum and the past-pointing will be absent.

In this case, then, we have complete loss of hearing in the left ear, with vertigo and entire failure of response on the part of the vestibular apparatus to the caloric reaction. There is no history of injury to the ear such as might have destroyed the function of the entire labyrinth. But even if a traumatism had occurred, the cochlear or hearing portion, being the more vulnerable, would have been lost first. If a systemic cause, such as a toxic condition due to nephritis, lues, alcohol, tobacco, etc., were present, both ears would be likely to be involved at the same time.

There is, however, a class of tumors, called by some "tumors of the *nervus acusticus*," situated in the cerebellopontine angle,

in which we have a certain peculiar symptomatic progress, with first labyrinthine symptoms, such as tinnitus, vertigo, loss of hearing, and diminished or lost vestibular response to caloric reaction; later there may be occipitofrontal pains, inco-ordination, and instability of cerebellar origin, involvement of adjacent cerebral nerves, then increased intracranial pressure, with choked disk, etc. Cushing says "that with definite cerebellopontine angle symptoms the diagnosis of an acoustic tumor is probable if characteristic labyrinthine responses to the caloric test are abolished, if deafness is complete, and if the *porus internus* shows a radiosopic enlargement." In our case no *x*-ray examination has as yet been made to see whether the internal auditory meatus is widened or not.

While we should hesitate in deciding definitely that this patient has a cerebellopontine angle tumor, we at least have several of the symptoms of the early stage of this disease present; namely, the total deafness of the ear, together with a complete loss of the reaction of the vestibular apparatus to the caloric test. Aside from the *x*-ray examination it will be necessary to observe this patient for a considerable time in order to see whether the other symptoms noted above will later occur in order to confirm this tentative diagnosis.

CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

IRREGULAR PLACEMENT AND FIXATION OF THE LARGE BOWEL

I HAVE recently had a group of rather interesting colon cases which bring out some suggestive findings in relation to irregular placement and fixation of the large bowel. The findings in most of these cases were entirely unlooked for and were disclosed in the routine general examination. However, in the majority of the cases these findings seemed to have a very direct relationship to the patient's condition and complaints, and improvement in these findings has benefited the clinical condition of the patients themselves.

The first patient, L. W., a merchant forty-four years of age, has complained of epigastric discomfort, constipation, offensive odor from the mouth, and "gas" for the past twenty years. He states that his trouble started, he believes, when he neglected to pay attention to the regularity of his bowel movements. The epigastric discomfort is described as a feeling of pressure, never amounting to true pain. He has quite severe eructations of gas. There is no vomiting, but occasionally slight nausea. Constipation is not habitual, but appears at irregular intervals, and he believes that when his bowels move regularly the symptoms are entirely relieved. He notices the offensive odor of his breath, particularly in the morning.

The balance of his history is negative and the physical examination throughout is negative, with the exception of some tenderness in the region of the gall-bladder and along the course of the transverse colon near the midline.

Laboratory examinations are without important significance, except the result of the opaque enema. The colon fills somewhat

slowly throughout. The hepatic flexure is held high under the right costal arch. The patient is tender at a point about 3 inches above and 2 inches to the right of the umbilicus. In the prone position the transverse colon takes a normal course (Fig. 45), but in the upright position the transverse colon appears to be hooked up to the hepatic flexure, which remains high (Fig. 46). The hepatic flexure has a limited range of mobility both in the upright and the prone positions.



Fig. 45.—Case I. x-Ray of barium-filled colon. Prone position.

The conclusions from this case, then, are that as a result of a probably chronic gall-bladder infection, with adhesions about the gall-bladder, the hepatic flexure has become fixed from pericolic adhesions, which, in the absence of all other physical and laboratory findings, is to be considered the factor of greatest importance in this patient. Although he has been under observation only three months, treatment directed to the loosening

up of these adhesions, together with general dietary and hygienic measures, has resulted in marked improvement in his bowel function, his epigastric distress, and his eructations of gas.

A somewhat similar group of symptoms is seen in this second patient, S. B., a gentleman sixty-four years of age, who for thirty-five years has complained of alternating periods of well-being and epigastric distress. This latter he describes as epigastric



Fig. 46.—Case I. Barium-filled colon. Upright position. Fixation of hepatic flexure in region of gall-bladder from pericolic adhesions.

pain coming on three or four hours after eating, and especially at night, which is relieved by the taking of food. It is frequently accompanied by eructations of gas and pyrosis, seldom by vomiting. There is no loss in weight.

His first attack, though mild, covered a period of one year and was followed by a remission of approximately five years. The second attack was approximately the same. Subsequent

attacks have been sharper and more severe, but of shorter duration. There is no history of tarry or clay-colored stools. There is no radiation of the pain.

The present attack started in February of this year and was accompanied by a severe sense of suffocation and chest oppression, and some dyspnea, which was more pronounced on lying down. Examination at that time revealed small areas of crepi-

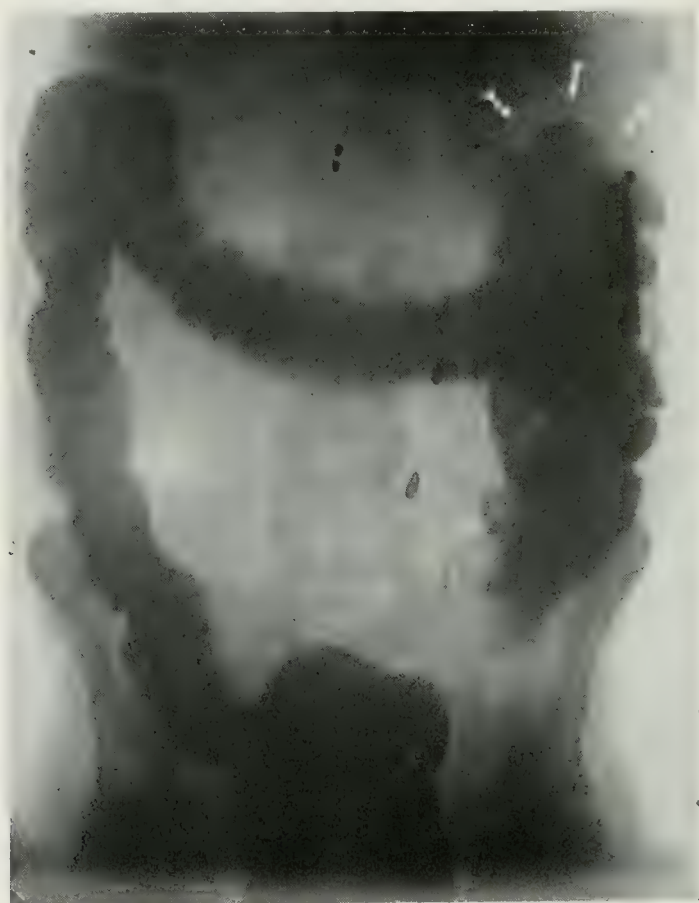


Fig. 47.—Case II. High position of hepatic flexure in prone position.

tant râles at both lung bases posteriorly, tenderness in the right upper quadrant, and a palpable, enlarged, tender liver edge.

Blood examination showed 4,400,000 red cells, 10,200 white cells. Blood-pressure, systolic 95, diastolic 55.

Urinalysis showed a trace of albumin, no sugar, no acetone, no casts, and a moderate number of white blood-cells.

Gastric analysis was negative.

α -Ray examination of the stomach was negative, except that the duodenum showed a limited range of mobility and was slightly irregular in outline.

The opaque enema showed that the colon fills slowly, but without defect. The hepatic flexure is held high under the right costal arch in the prone position (Fig. 47) and in the upright (Fig. 48).



Fig. 48.—Case II. Persistence of high position (fixation) of hepatic flexure in upright position.

The conclusion reached in this case is that Mr. B. suffers with a chronic cholecystitis of many years' duration, associated with pericolic and periduodenal adhesions. He has, in addition, a subacute fibrous myocarditis with heart muscle insufficiency. Because of this latter condition operation was not advised at the time of his entrance into the hospital. Following rest in

bed, digitalis, and regulation of his diet and bowel function he has made a complete symptomatic recovery. Within recent weeks he has been in the hands of an expert masseur and gymnast, and has been having abdominal massage, calisthenics, and bending exercises, with the result that the tenderness in his upper right quadrant, which had been present for years, as well as the general tendency to abdominal prominence, has been



Fig. 49.—Case III. Anomalous position of sigmoid colon, dilated, angulated splenic flexure. Prone.

markedly relieved, and the patient says he feels ten years younger. Although he has made a satisfactory recovery from this attack, one feels that if he has repeated recurrences of his gall-bladder trouble in the future surgical interference will be indicated.

The third case which I wish to present to you directs attention to the other side of the abdomen, showing the fixation of the de-

scending colon and splenic flexure in the left upper quadrant, as contrasted with the two previous cases in the right upper quadrant.

This patient, a gardener, thirty-three years of age, complains of headache, pyrosis, constipation, and gastric distress of four years' duration. His constipation is relieved by cathartics, but returns immediately after their use is discontinued. The



Fig. 50.—Case III. Dilated splenic flexure. Sigmoid colon held high and toward midline. Upright.

pyrosis frequently is associated with the feeling of a lump in the abdomen and is not very severe. It comes on about two or three hours after the noonday meal and is again relieved by taking food. He was in the Russian Army from 1908 to 1912, during which time, on coarse food and hard outdoor work, he felt entirely well.

Examination, both physical and laboratory, is negative

throughout, with the exception of the *x*-ray findings in the colon. The colon fills rapidly throughout. The sigmoid loop, instead of curving downward to the left in the left iliac fossa, takes an upward course toward the splenic flexure (Fig. 49). The splenic flexure itself is very large and out of proportion to the balance of the colon. It appears to be directed downward instead of upward in the normal location, and makes a very sharp turn upon itself. It appears as though the descending colon were held high and pulled upward, thereby pulling the sigmoid loop out of its usual location. The hepatic flexure is also held very high and maintains the same relation to the costal arch in both the upright and prone positions (Fig. 50).

The cause of this peculiar position and angulation of the splenic flexure could not be determined, but there seems little question that it is in some way related to his constipation. This patient has refused operation and so further information could not be obtained. Under general hygienic and dietetic care he has improved somewhat, but is still under treatment.

A later case, Case IV, M. H., a salesman forty-two years of age, suggests a somewhat similar condition, although the fluoroscopic examination after evacuation of the bismuth enema reveals a practically normal sigmoid and colon.

This patient has suffered from stomach distress, constipation, anorexia, pyrosis and eructations of gas for ten years. They come on periodically, usually two or three times a year. Between the attacks the patient is entirely comfortable.

A fractional meal showed a moderate hyperacidity and hypersecretion. He had at one time a positive Wassermann reaction, which, however, has become negative under intensive treatment, and has remained so up to the present time.

General physical examination is negative, as is also the laboratory examination. The opaque enema shows the colon to fill rapidly throughout. The sigmoid rises up to the splenic flexure, and it is impossible to bring it down (Fig. 51). Even in the upright position the sigmoid colon remains high. However, after evacuation of the colon contents the sigmoid comes down to the normal location (Fig. 52). It would seem that the



Fig. 51.— Case IV. Unusual position of sigmoid, rising directly to splenic flexure. Due to overfilling of barium enema.



Fig. 52.— Case IV. Normal position of sigmoid following evacuation of barium enema.

apparent unusual course of the sigmoid was probably due to pressure and overfilling of the barium enema.¹

In contrast to the above cases I wish to present very briefly 3 cases in women which show fixation in various unusual parts of the abdomen.

This first patient, Case V, a young married woman of thirty-two, complains of severe headaches and constipation. She has

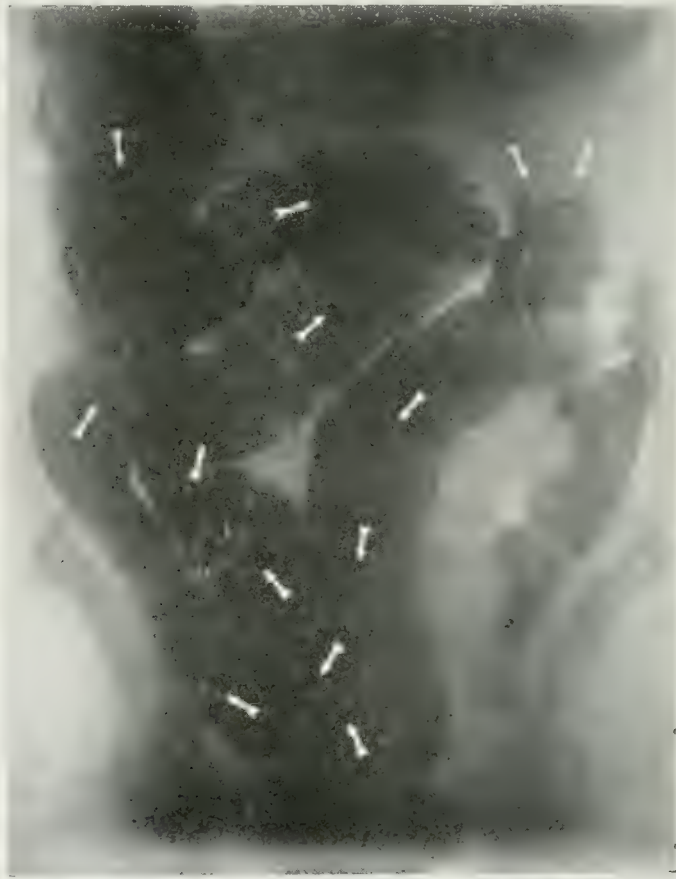


Fig. 53.—Case V. Obstinate constipation, cathartic colitis, redundant colon. Sigmoid fixed in right upper quadrant. Prone.

had various illnesses: fifteen years ago typhoid fever; eleven years ago appendectomy; three years ago tonsillectomy. She has had several miscarriages and several curetments. She is con-

¹ As a result of the experience with these cases patients are always examined after evacuation of the barium mass in order that any artefact result of the filling may be ruled out.

stipated and is addicted to the constant use of various cathartics. When severely constipated her headaches are very much worse.

The barium enema showed that the sigmoid colon instead of taking its usual course to the left runs straight from the ampulla upward to the right, under the right costal arch, in close relationship to the hepatic flexure (Fig. 53). In the upright position both the hepatic flexure and the sigmoid colon remain fixed (Fig. 54).



Fig. 54.—Case V. Fixation of sigmoid and hepatic flexure in right upper quadrant. Upright.

One cannot help feeling from the appearance of this colon, with its fixation and its many angulations, that such a case would probably be markedly benefited by resection of the redundant colon and freeing of the immobilized areas. This patient, like many others with similar complaints, refuses surgical operation, and in spite of various methods of medical treat-

ment continues to take ever-increasing amounts of cathartics to insure bowel movements.

The next patient, Case VI, a woman forty-two years of age, suffering from a severe colitis, accompanied by abdominal pain and constipation, shows a fixation in the left lower quadrant of her abdomen (Fig. 55). As you will see, the transverse colon as



Fig. 55.—Case VI. Fixation of transverse, ascending, and descending colon in left lower quadrant, occurring in case of severe colitis, due to old adhesions around left adnexa.

well as the ascending portion is fixed down in the pelvis. There is a very limited range of mobility and the patient is likewise tender over this area. On attempting to bring this fixed portion out of the pelvis the patient was placed in the Trendelenburg position, but without success (Fig. 56).

You will note that this picture has not been copied the same

way that the others have, so you must picture to yourselves that the position is on the left side, the same as it is seen in Fig. 55. These left-sided adhesions are probably associated with an old inflammatory process of the left adnexa, for which the patient has been under treatment for several years. Operation has not been considered in this case because of her desire to go through a period of six months with active exercise, particularly golf,



Fig. 56.—Case VI. Trendelenburg position, in attempt to lift fixed bowel out of pelvis.

abdominal massage, calisthenics, and olive oil injections. This far, in a period of four months, she has been making very good progress, although my own feeling is that sooner or later she will consent to operative interference.

This last patient, Case VII, Mrs. F., a widow, fifty-nine years of age, with a mild Bright's disease associated with hyperarterial tension and slight heart muscle insufficiency, shows a fixation of

the transverse colon to a point in the midpelvis, probably to the pouch of Douglas and the dome of the uterus.

The report of the opaque enema is as follows: The colon fills slowly, the sigmoid fails to rise out of the pelvis, the transverse colon drops sharply down from the splenic flexure to the ampulla, and then back up again to the hepatic flexure. The



Fig. 57.—Case VII. Fixation of transverse colon to ampulla of rectum.

transverse colon cannot be separated from the ampulla and the patient is exquisitely tender over this point (Fig. 57).

Because of this patient's cardiorenal condition it does not seem advisable to institute operative procedure for relief. She has been advised to have treatment directed toward the reduction of blood-pressure, which is around 180 systolic, and in the event that her condition improves she will be advised to have this condition of the colon relieved by proper surgical measures.

This group of cases illustrates the value and importance of colon study in the elucidation of a rather obscure group of chronic gastro-intestinal patients. Of course, such study should be part of the general complete examination, and only after organic disease in other organs has been ruled out by the usual laboratory and clinical examinations should reliance be placed on these abdominal findings. Further, before too much importance is given to them, one must familiarize himself with the



Fig. 58.—Normal colon. Recumbent (prone) position.

normal colon in order that the degrees of mobility and the position of the bowel in relation to other organs are well understood.

These plates (Figs. 58, 59) illustrate a normal colon filled with a barium enema in the prone and upright positions.

I hope that this little talk may stimulate some of you to engage in such studies, for in recent years while much careful work has been done on the stomach, duodenum, gall-bladder, and kidney in *x*-rays, the colon has been a somewhat neglected field, particularly in cases of this type, where gross pathology is wanting. Those of you who are interested in such studies would do well

to read a most complete study of the colon recently published by Case in the *Americal Journal of Roentgenology*, Vol. IV, August 17th, No. 8, pp. 373-388, and called "The Pelvic Colon and Rectum Roentgenographically Considered."

If one be permitted to summarize briefly the complaints of these patients suffering with irregular placement and fixation of the colon, they might be stated as follows:



Fig. 59.—Normal colon in upright position. Note fixation of splenic flexure (normal) and descent of hepatic flexure (normal) on change of posture.

Most of these patients are *chronic* sufferers—their complaints covering a period of many years—in this series from four to thirty years. During this period their complaints have been frequent exacerbations and remissions, but with no regularity in their appearance. Of these complaints, *severe eructation of gas*, *pyrosis*, slight nausea, anorexia, and *constipation* are strikingly and rather uniformly found. Headache, indefinite gastric distress occurring after meals, easy tire, and fatigue are also frequently noted. Objectively, these patients appear rather sallow, with muddy skins, rather heavy and lethargic, tired, and

for the most part depressed. Not infrequently evidence of an old abdominal inflammation may be elicited, cholecystitis, splenitis, perisplenitis, appendicitis, salpingitis, ovaritis, etc., etc., which infections probably have intimate etiologic relationship with the production of these colon adhesions and fixation. Also the older of these patients show evidence of cardiac or cardiorenal involvement, probably the end-result of those earlier abdominal infections.

CLINIC OF DR. FRANK WRIGHT

MICHAEL REESE HOSPITAL

A CONSIDERATION OF THE ABNORMAL LOSS OF FLUID IN CONTRAST WITH EDEMA

THE fact that the excretion of salt by a patient placed upon a salt-poor diet is accompanied by the loss of fluid from the tissues is one taken advantage of frequently to bring about a reduction of edema. It is comparatively infrequent that one meets the clinical condition in which the converse is true, one in which large quantities of solid lost through the kidney have resulted in the excretion of so much fluid that the patient shows marked evidence of desiccation.

For this reason the following patient is presented, particularly in view of the fact that he added 25 per cent. to his body weight in a month's time, developed a slight edema, and in the process of ridding himself of this edema brought out some interesting figures (see table, p. 274):

G. T. Michael Reese Hospital. No. 8157. Forty-three years of age. Tailor.

In May, 1916, he joined the Canadian forces, infantry division, landing in England in October, and in France in December, 1916. He was sent to the front and fought until June, 1917, when he noticed that he had lost some weight and did not feel as strong as he did previously. He reported sick to the battalion doctor, who thought the patient was suffering from rheumatism, but after treatment with no improvement the patient was sent to a base hospital at Boulogne. At the end of a week, feeling better, he was returned to the front. He remained at the front about a month, but found he could not

"carry on" and was sent back to Boulogne. Until December, 1917, he felt pretty well, and worked about the hospital. As yet no test of the urine was made, but in December urine examination revealed the presence of sugar and the diagnosis of diabetes was made.

After this he was sent to a military hospital in London, put on a rather rigid diet for his diabetes until March, 1918, and became sugar free. From March until July he was in a Canadian convalescent home in Berkshire, England, where no attention was paid to his diet. He was sent to Liverpool early in July, and arrived in Canada the latter part of July, 1918. Upon reaching his home, Winnipeg, he consulted his physician, who found something over 100 grams of sugar in the day's urine, put him upon a non-carbohydrate diet, which was followed only partially, but resulted in his feeling much stronger than he did previously. In December, 1918, he began his work as a tailor, disregarding the advice of the physician. Things progressed so well during January and February of that year that he reached the conclusion he was cured, and that he would indulge his own inclinations regarding diet. There followed then an orgy of pastry and candy eating which is hard to describe. Coupled with the soldiers' liking for sweets was the memory of the ban placed upon them by his physician, and as soon as that was forgotten he ate his fill of cake, ice-cream, mince pie, candy, and chocolate bars. In a few days he noticed a loss of strength. First, a rest of a few minutes would allow him to resume his occupation, that of a tailor. He lost weight rapidly, his appetite failed, and enormous thirst developed in the course of a few days, and shortly afterward a polyuria, neither of which had been noted at any time before. March 20th he was forced to quit work and go to bed, and ten days later he left for Chicago to enter Michael Reese Hospital.

In May, 1916, he weighed 178 pounds. Twenty-one months ago, on being sent to a hospital in France, he weighed 154 pounds, and upon entrance here he weighed $101\frac{3}{4}$ pounds, having lost over 75 pounds in three years; fully 40 pounds being lost in the three months previous to entrance.

Upon entrance the most striking thing noted was the wrinkled, ape-like appearance of the face, and with it a decided pigmentation of the exposed parts, less marked on the surface covered by the clothes. The skin was dry and the subcutaneous fat almost lacking, yet the appearance did not simulate the cachexia of malignancy. The muscles were small and flabby. Physical examination revealed little but a systolic blow over the base of the heart. No organs palpable in the abdomen; all reflexes present.

The first day in the hospital, April 3, 1919, the quantity of urine was 3330 c.c., sp. gr. 1027, dextrose 126 gm., ammonia 2.4 gm., albumin a trace; acetone; marked diacetic acid present, no casts found.

The blood showed: Hbg. 95 per cent.; W. B. C. 7200; Differential: (1) P. N. 58, (2) Lymph. 32, (3) Lg. Mon. 8, (4) Eos. 2. Wassermann negative. Blood-pressure: Systolic, 102; diastolic, 82.

Because of weakness it was thought best to institute a rather full diet and gradually reduce it, meanwhile keeping a strict watch for any untoward symptoms.

Beginning with 120 grams of carbohydrate and 120 of fat, a total of 2500 calories, the patient excreted 216 grams of sugar, the urine showing large amounts of acetone and diacetic acid, with 2.4 grams of ammonia. The amounts were gradually reduced until two weeks later a diet of 18 gm. carbohydrate, with 75 gm. fat, with a total calories of 1020, resulted in but 46 gm. of sugar excreted. The diacetic acid appeared in traces only, and the patient had gained 7 pounds in weight.

Starvation days were then instituted every seventh or eighth day, and the result was a prompt disappearance of the diacetic acid, and reductions in the urinary ammonia; after the second starvation the urine failed to show sugar for the first time. His tolerance to carbohydrate was practically nil, however, and two days after the starvation the urinary dextrose approximated in amount the quantity of carbohydrate ingested.

In the meantime the patient had considerable thirst, and took water freely, his weight increasing to $126\frac{3}{8}$ pounds thirty-

Date.	Weight, pounds.	Twenty-four-hour Urine				Food						Blood		
		Quantity, c.c.	Salt.	Sugar.	Diacetic acid.	Liquid.	Salt.	Carbo- hydrate.	Protein.	Fat.	Calories.	Sugar, %	Urea, mgm. per 100 c.c.	CO ₂ vol. %
Apr. 3	101 $\frac{3}{4}$				++									
4		3330		126	++			123	120	121	2432			
5		3930		216	+			120	131	141	2480			
6	101 $\frac{1}{4}$	4140		172	+			121	141	143	2344			
7		3840		192	+			69	111	122	1820			
8	102 $\frac{1}{8}$	3600		165	++			70	94	142	1920	0.24	34	44
9		3030		120	++			51	92	114	1600			
10	105 $\frac{1}{4}$	3210		133	+			48	93	104	1500			
11		4620		143	++			30	67	105	1330			
12	104 $\frac{3}{8}$	3780		114	++			25	67	96	1230			
13		3240		112	+			20	64	71	1050			
14	105 $\frac{1}{2}$	2920		67	+			18	65	97	1200			
15		2880		53	Trace			17	76	85	1130			
16	107 $\frac{1}{8}$	2880		65	Trace			18	68	80	1060			
17		2300		44	Trace			19	62	69	1025			
18	108 $\frac{1}{8}$	2340		42	Trace			18	67	75	1020	0.18	36	48
19		2240		46	+			Starvation						
20	109 $\frac{1}{8}$	3840		20	0			11	22	96	1070			
21		3840		14	0			11	22	97	1080			
22	115 $\frac{3}{4}$	4440		11	0	3360	24	15	35	108	1260			
23	117 $\frac{1}{8}$	5840		17	0	3220	24	17	52	113	1370			
24	118 $\frac{1}{8}$	5580	24	30	0	4020	25	19	55	162	1752			
25	120 $\frac{1}{8}$	4020	23	23	0	4680	25	20	54	169	1820			
26	119 $\frac{1}{8}$	4920	25	32	0	4320	20	Starvation						
27	118 $\frac{1}{8}$	4020	14	0	0	3000	25	12	23	107	1100			
28	121 $\frac{1}{2}$	1980	14	Trace	0	3000	25	12	32	131	1350			
29	122 $\frac{1}{8}$	3240	24	0	0	3780	25	13	39	130	1360	0.18	25	54
30	122 $\frac{1}{8}$	3720	30	12	0	3240	25	15	44	128	1470			
May 1	123	3240	19	12	0	3450	25	14	47	134	1450			
2	124 $\frac{1}{8}$	2520	19	Trace	0	5970	25	Starvation						
3	124 $\frac{1}{8}$	5880	28	13	0	3450	25	13	28	65	750			
4	125 $\frac{1}{8}$	3420	29	0	0	4140	25	13	38	78	900			
5	126 $\frac{1}{8}$	3720	27	0	0	3420	24	14	47	90	1050			
6	125 $\frac{1}{4}$	3000	30	Trace	0	2500	10	21	60	127	1460			
7	124 $\frac{1}{8}$	2100	20	0	0	2500	10	22	73	122	1470			
8	122 $\frac{2}{8}$	2880	23	12	0	2500	10	23	67	127	1500			
9	119 $\frac{6}{8}$	3720	24	22	0	3030	15	25	67	176	1950			
10	118 $\frac{1}{8}$	3700	22	29	0	3000	15	25	66	153	1747	0.18	25	50
11	116	4620	31	37	0	3000	15	22	66	154	1820			
12	114 $\frac{1}{2}$	3300	26	42	0	4000	15	Starvation						
13	113 $\frac{1}{8}$	3900	23	14	0	4500	20	22	65	155	1820			
14	114 $\frac{1}{8}$	3360	20	12	0	4500	25	20	66	130	1700			
15	116 $\frac{1}{4}$	4140	26	20	0	4920	25	30	88	197	2250			
16	114 $\frac{3}{4}$	5640	38	55	0	5310	25	Starvation						
17	113 $\frac{1}{8}$	3840	25	18	0	2010	15	30	93	200	2450			
18	113 $\frac{7}{8}$	2040	22	37	0	4020	25	31	93	203	2480			
19	115 $\frac{1}{8}$	3720	33	48	0	3570	25	15	75	172	2080			
20	114 $\frac{3}{8}$	4140	33	52	0	5340	25	Starvation						
21	115 $\frac{1}{8}$	4140	35	19	0	3540	25	15	88	216	2518			
22	117 $\frac{3}{8}$	2520	25	20	Trace	3750	25	15	80	210	2440			
23	116 $\frac{1}{2}$	4200	27	35	0	3060	25	16	82	206	2410			
24	115 $\frac{1}{4}$	5520		66	0	4860	25	14	82	211	2445			
25	112 $\frac{1}{4}$	3960	31	36	0	3240	25	14	80	203	2360	0.14	23	54
26	112 $\frac{1}{8}$	2550	16	46	0	3240	25	Starvation						
27	115 $\frac{1}{8}$	2160	16	19	0	4100	25	16	79	170	1910			
28	116 $\frac{1}{4}$	2310	19	11	Trace	4470	25	15	82	122	2176			
29	119 $\frac{1}{4}$	3550	26	19	0	2940	25	Starvation						
30	120 $\frac{5}{8}$	3155	25	15	0	5160	25	16	78	183	2020			
31	122 $\frac{3}{8}$	1525	10	0	0	3150	25	16	92	182	2230			
June 1	125 $\frac{5}{8}$	1500	17	5	0	3030								
2	127 $\frac{7}{8}$	2000		21	0	2800								

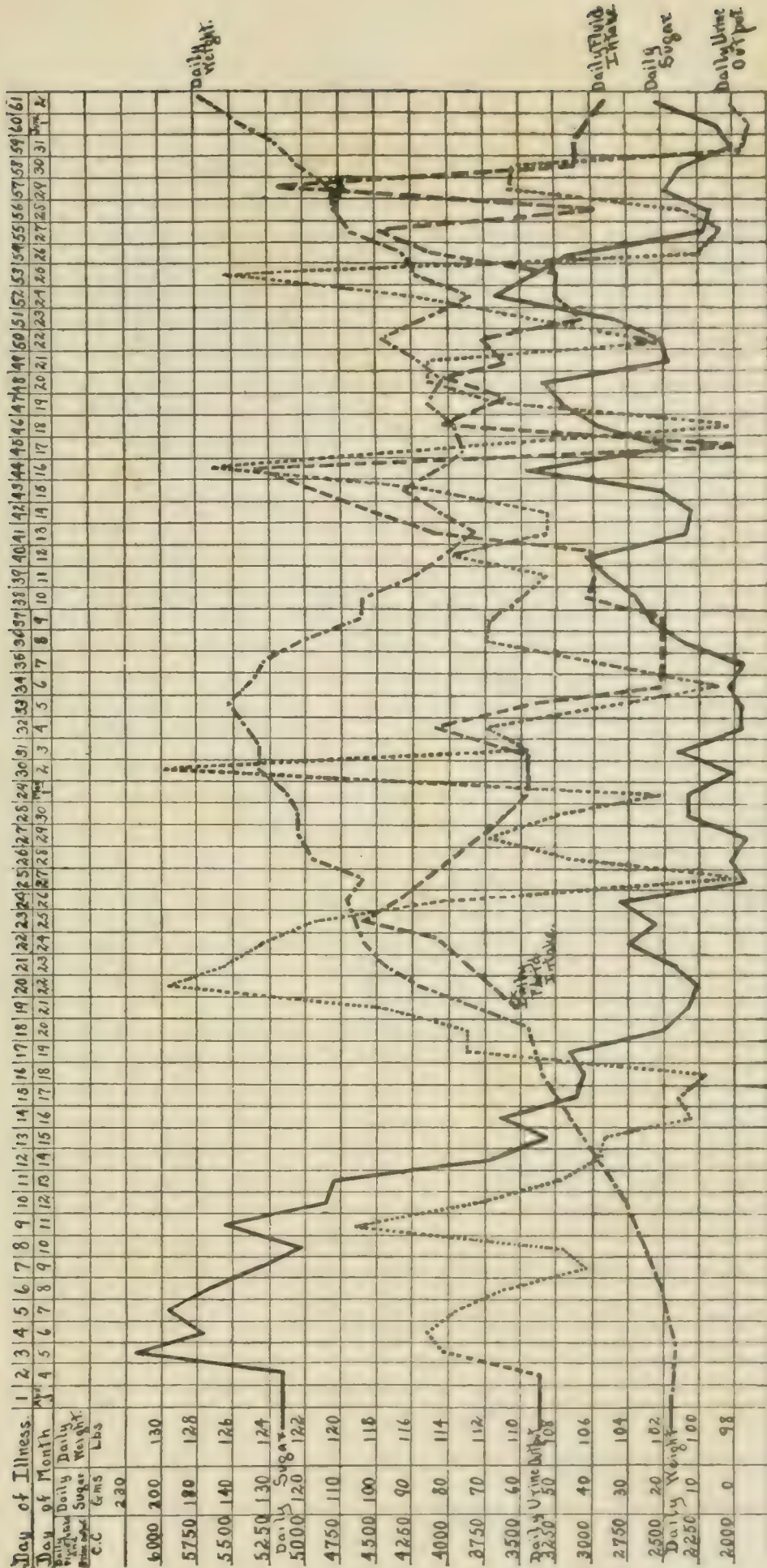


Fig. 60.—Chart showing curves referred to in text.

two days after entrance, a gain of 25 pounds. On this day an edema of the feet and ankles appeared suddenly.

The salt was at once reduced in amount and the edema disappeared in three days, but the weight continued to fall until there was a loss of 13 pounds in eight days. At this time the carbohydrate was given in amounts varying from 20 to 25 gm., and calcium glycerophosphate administered in 2-gm. doses daily. There was considerable excess in urine output as contrasted with fluid intake, and the dextrose in the urine exceeding the carbohydrate intake.

The change in weight varied in inverse proportion to the dextrose output in the urine, and the day following the steady decline in the amounts of sugar excreted, the figures for weight mounted steadily until the loss of 13 pounds was regained. This is shown graphically in the chart, the weight curve being practically a mirror-like replica of the sugar output curve.

If asked why the gain in weight is encouraged, I can quickly answer, The patient feels much better when at the higher weight.

There are a number of features which make this case interesting to me: First, being the onset at a time when the patient was subjected to the strain of fighting, and at the same time eating all the sweets he could obtain; next the breaking down of his carbohydrate tolerance by eating excessively of sweets when he had been feeling fairly well; then the desiccation and the marked inverse relationship between the sugar output and the weight; the production of the edema; the pigmentation suggesting endocrine gland involvement; the decided tolerance for fats, and disappearance of acidosis while being fed large quantities of fats; the loss of strength quickly following the reappearance of dextrose in the urine, with the concomitant loss in weight, and the remarkable gain in strength and feeling of well being, which follows the gain in weight even before the sugar has disappeared.

I will make no attempt to interpret the blood findings, for in discussing the case with my chief, Dr. Chester Farmer, he asked me "Why didn't you watch the specific gravity of the blood to see if it changed, and so get some idea of what influence

this water absorption has upon the body fluids?" That is an oversight I hope to remedy.

At present there is practically no sugar tolerance, and in view of the patient's disinclination to follow a strict diet I fear that even if he becomes able to assimilate fair amounts of carbohydrate, he may break bounds and quickly destroy such little tolerance as he may so laboriously build up.

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